

## The Covariation of Trait Anger and Borderline Personality: A Bivariate Twin-Siblings Study

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Anger can be defined as an emotion consisting of feelings of variable intensity, from mild irritation or annoyance to intense fury and rage. Borderline personality disorder (BPD) is characterized by impulsivity and instability of interpersonal relationships, of self-image, and of negative affects. Borderline personality and trait anger are often observed together. The present study examined the extent to which a genetic association explains the covariation between a trait measure of borderline personality and trait anger. To this end, self-report data of 5,457 twins and 1,487 of their siblings registered with the Netherlands Twin Register and the East Flanders Prospective Twin Survey were analyzed using genetic structural equation modeling. A significant phenotypic correlation was observed between the two traits ( $r_P = .52$ ). This correlation was explained by genetic (54%) and by environmental influences (46%). A shared genetic risk factor is thus one of the explanations for the covariation of borderline personality and trait anger.

*Keywords:* borderline personality, trait anger, twin study, genetic factors

Anger can be defined as an emotion that consists of feelings of variable intensity, from mild irritation or annoyance to intense fury and rage (Spielberger, Jacobs, Russell, & Crane, 1983). It can be conceptualized as *state anger*, referring to an episode of anger occurring at a specified time, or as *trait anger*, referring to an aspect of personality (Eckhardt, Norlander, & Deffenbacher, 2004). Feelings of anger may be suppressed or directed inward

(“anger-in”) or expressed outwardly in some form of aggressive behavior (“anger-out”). Anger has been related to several important constructs in behavioral medicine and psychological research. For example, high levels of internal expression of anger and trait anger have been associated with increased blood pressure and induced hypertension (Markovitz, Matthews, Wing, Kuller, & Meilahn, 1991; Schneider, Egan, Johnson, Drobny, & Julius, 1986), increased risk for coronary heart diseases (Atchison & Condon, 1993; Williams et al., 2000; Leon, 1992; Kawachi, Sparrow, Spiro, Vokonas, & Weiss, 1996; Eaker, Sullivan, Kelly-Hayes, D’Agonstino, & Benjamin, 2004; Chang, Ford, Meoni, Wang, & Klag, 2002), and mental disorders such as anorexia and bulimia nervosa (Fassino, Daga, Piero, Leombruni, & Rovera, 2001) bipolar disorder (Posternak & Zimmerman, 2002), and borderline personality disorder (Morse et al., 2009).

Borderline personality disorder (BPD) is characterized by a pervasive pattern of instability of interpersonal relationships, of self-image, and of negative affects, and marked impulsivity that begins by early adulthood and is present in a variety of contexts (American Psychiatric Association [APA], 2000). BPD is diagnosed in approximately 1% to 2% of the general population (Lenzenweger, Lane, Loranger, & Kessler, 2007; Torgersen, Kringlen, & Cramer, 2001) and is associated with a variety of negative outcomes such as self-harm behavior, suicidal behavior, impaired occupational and interpersonal functioning, delinquent behavior, and substance abuse (Skodol et al., 2002). Inappropriate, intense anger or difficulty controlling anger is the most prevalent BPD criterion in clinical samples (Zanarini, Frankenburg, Hennen, Reich, & Silk, 2005), nonclinical samples (Trull, 1995), and in first degree relatives of BPD patients (Zanarini et al., 2004). Further,

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this feature was found to be among the slowest symptoms to remit in BPD patients across 8- to 10-year follow up (Zanarini et al., 2007).

Both trait anger and the trait of borderline personality are heritable. Rebollo and Boomsma (2006) conducted a longitudinal twin family study into the genetics of trait anger. The genetic architecture of the trait differed in men and women. In males 23% of the variance was explained by additive genetic effects and 26% by dominant genetic effects, leading to a total heritability of 49%. In women, 34% of the variance was explained by additive genetic effects and no dominant genetic effects were found. Other studies only focused indirectly on the heritability of anger or violence, for example in genetic studies on antisocial personality disorder for which the heritability was estimated at 38% (Cadoret & Stewart, 1991; Torgersen et al., 2008). Large-scale twin and twin family studies of BPD and the trait of borderline personality report heritability estimates around 40% (Distel et al., 2008a; Bornovalova et al., 2009; Kendler et al., 2008; Torgersen et al., 2008; Distel et al., 2009). The study by Distel et al. (2009), included twins as well as their parents and nontwin siblings and provided evidence for the influence of nonadditive genetic effects, while suggesting no effects of cultural transmission from parents to offspring.

Some overlap between trait anger and the trait of borderline personality is expected, considering that one of the nine criteria for BPD concerns inappropriate expressions of anger and intense, chronic feeling of anger (APA, 2000). However, most of the BPD criteria do not directly tap trait anger. On the other hand, there are many cognitive processes associated with trait anger (Owen, 2011; Wilkowski & Robinson, 2010) that may at least partially explain some of the remaining symptoms and features of borderline personality. For example, selective attention and reasoning biases associated with trait anger may lead individuals to be hypervigilant to possible threat or aggression and to attribute hostile intentions to others so as to arouse anger more frequently (Wilkowski & Robinson, 2010). These cognitive processes associated with anger may lead to rejection sensitivity and to interpersonal conflict and disruption often seen in those with BPD, for example (Romero-Canyas et al., 2010). Therefore, an examination of the phenotypic and genotypic association between trait anger and borderline personality can help to index the degree to which the two traits may share a common underlying cause as well as point to potential shared mechanisms (e.g., cognitive processes and biases) that may inform theories of the etiology of BPD.

In the present study, we explored shared genetic risk factors as a possible explanation for the covariation of borderline personality and trait anger in the population. Data from twins and their siblings were available from the Netherlands Twin Register (NTR; Boomsma et al., 2006) and the East Flanders Prospective Twin Survey (EFPTS; Derom et al., 2006) to disentangle genetic and environmental influences on the covariance between borderline personality and trait anger.

## Methods

### Participants

The present study is part of an ongoing study on health, lifestyle, and personality in twins and their family members registered with

the NTR established in 1978 (Boomsma et al., 2006). Every 2 years, surveys on health and lifestyle were sent to the twin families. For the present study, data from the seventh survey were used which was sent in 2004–2005. Dutch-speaking twins in Belgium were also asked to take part in the Dutch health, lifestyle, and personality study. Belgian participants were recruited through the EFPTS, a population-based register of multiple births in the Belgian province of East Flanders which was started in 1964 (Derom et al., 2006). Young adult twins were contacted by mail and invited to complete a survey which was enclosed with the letter. A nonresponse study (Distel et al., 2007) showed that for a substantial group of targeted participants the addresses were incorrect. This group thus never received the questionnaire. After correcting for this, the response rate was estimated at 52.2% in the group of targeted participants who participated before and 13.6% in the group of targeted participants who were already registered, but never completed a questionnaire.

For the Dutch sample zygosity was determined either from DNA typing or from self-report answers to eight survey questions on physical twin resemblance and confusion of the twins by family members and strangers. Zygosity agreement reached 97% (Willemsen, Posthuma, & Boomsma, 2005). For the Belgian sample, twin zygosity was determined through sequential analysis based on sex, fetal membranes, umbilical cord blood groups, and placental alkaline phosphatase until 1985. After that time, DNA fingerprinting was used. In case of missing or insufficient DNA information, the zygosity of the same-sex DZ twins was based on survey items on physical twin resemblance and confusion of the twins (see Derom & Derom, 2005).

Data from 7,261 twins and siblings with valid scores on the trait measures of borderline personality and anger were available. A total of 928 twins were registered with the EFPTS. Twins with unknown zygosity ( $N = 94$ ), individuals with an unknown age ( $N = 148$ ) or sex ( $N = 12$ ) and individuals aged below 18 ( $N = 14$ ) were excluded. A maximum of two brothers and two sisters were included in the analyses, remaining siblings were excluded ( $N = 49$ ). This resulted in a total sample of 5,457 twins and 1,487 siblings from 3,946 families. The twin sample consisted of 813 monozygotic males (MZM), 416 dizygotic males (DZM), 2,095 monozygotic females (MZF), 1,008 dizygotic females (DZF), 1,125 dizygotic opposite sex (DOS), and 528 brothers and 959 sisters. Table 1 shows the complete family configuration of the sample. There were 1,866 families in which both members of a twin pair completed the questionnaire, 1,725 families in which only one member of the twin pair completed the questionnaire and 355 families in which only nontwin siblings completed the questionnaire. The mean ( $M$ ) age of the twins was 34.46 years (standard deviation [ $SD$ ] = 10.98, range = 18–87 years). The mean age for the siblings was 39.89 years ( $SD = 12.55$ , range = 18–91 years).

### Measures

Trait anger was measured with the Dutch adaptation of the State Trait Anger Scale (STAS, Spielberger et al., 1983; van der Ploeg, Defanes, & Spielberger, 1982). The scale is designed to assess the frequency of which an individual experiences the state anger over time and in response to a variety of situations. The STAS-adaptation was scored on a 4-point Likert scale (1–4; almost never,

Table 1

*Family Configuration in the Sample According to Zygosity, Cohort, and Number of Additional Nontwin Siblings*

	Families yielding					Total
	No siblings	1 sibling	2 siblings	3 siblings	4 siblings	
<b>MZM</b>						
Families yielding a twin pair	202	64	12	6	1	285
Families yielding a single twin	207	27	9	0	0	243
<b>DZM</b>						
Families yielding a twin pair	67	35	3	3	1	109
Families yielding a single twin	157	35	6	0	0	198
<b>MZF</b>						
Families yielding a twin pair	613	164	39	9	2	827
Families yielding a single twin	373	56	12	0	0	441
<b>DZF</b>						
Families yielding a twin pair	233	70	24	2	0	329
Families yielding a single twin	290	42	14	4	0	350
<b>DOS</b>						
Families yielding a twin pair	223	79	11	2	1	316
Families yielding a single twin	404	67	17	4	1	493
Families yielding no twins	—	282	62	10	1	355
<b>Total</b>	<b>2769</b>	<b>921</b>	<b>209</b>	<b>40</b>	<b>7</b>	<b>3,946</b>

Note. MZM = monozygotic males; DZM = dizygotic males; MZF = monozygotic females; DZF = dizygotic females; DOS = dizygotic opposite sex.

sometimes, often, almost always) and consists of 10 items concerning for example getting easily annoyed and irritated, being a hotheaded person, flying off the handle, and having a fiery temper. Participants were asked to indicate the extent to which an item occurred in their everyday lives. The items were scored according to the test manual, which states that at least 80% of the items must be answered to calculate a sum score and that missing values or ambiguous answers should be substituted by a score of 2 (sometimes true). Several studies have reported excellent psychometric qualities, reliability ( $\alpha$ ) of 0.86 and good discriminant and convergent validity (Eckhardt et al., 2004). In the present sample, the internal consistency (Cronbach's alpha) was 0.85. Borderline personality was measured by the Dutch translation of the Personality Assessment Inventory – Borderline features scale (PAI-BOR; Morey, 1991). The PAI-BOR consists of 24 items rated on a 4-point scale (0–3; false, slightly true, mainly true, very true) that tap features of severe personality pathology clinically associated with BPD, such as stability of mood and affects, self-image, feelings of emptiness, intense and unstable relationships, impulsivity, and self-harm. The items were scored according to Morey's test manual, which states that at least 80% of the items must be answered to calculate a sum score and that missing values or ambiguous answers should be substituted by a zero score. Several studies in clinical, as well as nonclinical samples, have supported the reliability and validity of the PAI-BOR total score in indexing the degree to which borderline features are present (Distel et al., 2008b; Trull, 1995; Trull, 2001; Morey, 1991). Bell-Pringle et al. (1997) and Stein, Pinkster-Aspen, and Hilsenroth (2007), for example, showed that the PAI-BOR differentiates between patients diagnosed with BPD and patients without borderline personality pathology or unscreened controls with 75% to 80% accuracy. Jacobo, Blais, Baity, and Harley (2007) administered the PAI-BOR to patients diagnosed with BPD and found a significant correlation of .58 between the total number of BPD SCID-II criteria and the PAI-BOR scale. Finally, the 6-month test–retest

correlation of the Dutch version of the PAI-BOR assessed on 200 unrelated individuals was 0.78 (Distel et al., 2008a) and multi-group confirmatory factor analysis showed that the PAI-BOR is measurement invariant across sex and age (De Moor, Distel, Trull, & Boomsma, 2009).

The PAI-BOR does include two items that directly assess anger, one asking whether the respondent has control over his or her anger, and the other asking whether the respondent gets so mad, s/he has trouble controlling feelings of anger. Therefore, as detailed below, we repeated all phenotypic and genotypic analyses after deleting these two PAI-BOR items from the total PAI-BOR score. In the present sample, the internal consistency (Cronbach's alpha) was 0.83 for the full PAI-BOR scale and 0.82 for the 22-item scale.

## Twin Studies

In twin-family studies, the different degree of genetic relatedness of monozygotic (MZ) and dizygotic (DZ) twins and sibling pairs is used to identify the relative contribution of genes and environment to the phenotypic variation in a trait, or to the covariation between traits. MZ twins are (almost) genetically identical and DZ twin and sibling pairs share on average half of their segregating genes (Boomsma, Busjahn, & Peltonen, 2002). The total phenotypic variance is decomposed into a part due to the additive effects of alleles at all genomic loci (A), the nonadditive genetic effects of alleles (D; dominance), the effects of the environment that is shared by individuals growing up in the same family (C), and the effects of nonshared environment (which also includes measurement error, E). The expectation for the phenotypic variance may be written as:  $V_{(P)} = V_{(A)} + V_{(D)} + V_{(C)} + V_{(E)}$ . Broad-sense heritability ( $h^2$ ) is the proportion of phenotypic variance that is attributable to genotypic variance,  $h^2 = [V_{(A)} + V_{(D)}]/V_{(P)}$ ; narrow-sense heritability is the proportion of variation explained by additive genetic factors,  $h_n^2 = V_{(A)}/V_{(P)}$ . Based on

data from only MZ and DZ twins and siblings this full model (i.e., ACDE) is not identified, and either an ADE or an ACE model can be fitted to the data. The choice between these latter two models may be based on prior knowledge or on the pattern of correlations in MZ and DZ twins. When the DZ correlation is more than half the MZ correlation, there is evidence for environmental effects shared by twins from the same family (C) and when the DZ correlation is less than half the MZ correlation, there is evidence for nonadditive genetic effects (D). In the present study an ADE model was fitted to the data. The ADE model is identified because of the difference in correlations among the latent factors influencing the phenotype in MZ and DZ twin pairs. For MZ twin pairs correlations between the A and the D factor score in twin 1 and twin 2 are both one. For DZ and sibling pairs, these correlations are 0.5 and 0.25, respectively. Correlations between unique environmental factor scores in twin 1 and twin 2 are zero in MZ and DZ pairs (e.g., Falconer & Mackay, 1996; Boomsma & Molenaar, 1986).

Bivariate genetic analyses can be applied to determine to what extent the covariation between two traits can be explained by genetic and environmental factors. The comparison of MZ and DZ cross-twin cross-trait correlations provides a first indication about the shared etiology between traits. If a significant cross-twin cross-trait correlation is present it suggests that there is a familial influence on the etiology of the correlation between the two traits. If the MZ cross-twin cross-trait correlation exceeds the DZ cross-twin cross-trait correlation it suggests that the familial influence on the correlation is at least partly genetic in origin. A graphical representation of the bivariate genetic model is shown in Figure 1.

**Statistical Analyses**

We first fitted a saturated model in which variances, covariances (among family members and among traits) and means were estimated. Mean borderline personality and trait anger scores were

estimated separately for twins and siblings. An effect of sex (coded as 0 for males and 1 for females) and age (in years) were included as fixed effects (regression coefficients) on each trait.

We tested for the significance of differences in mean scores of twins and siblings and for the effect of sex and age on borderline personality and trait anger scores. Significant effects of sex and age were retained in subsequent genetic analyses. All correlations between MZ and DZ twin and sibling pairs within and between traits were estimated as a function of zygosity and sex. By constraining within-trait and cross-trait correlations to be equal for DZ twins and nontwin siblings and between men and women within the zygosity groups we tested for a specific twin environment and for qualitative and quantitative sex differences. Qualitative sex differences (i.e., different genes influence the trait in males and females) are suggested if correlations in DZ twins of opposite sex (DOS) cannot be predicted based on the pattern of correlations in same-sex twin pairs. Quantitative sex differences (differences in the magnitude of A, D, and E between males and females) are suggested when the correlations in male–male and female–female pairs within zygosity cannot be constrained to be equal without a significant deterioration in the fit of the model.

To assess to what extent borderline personality and trait anger share genetic liability, a bivariate genetic model was fitted to the data in which the variance in borderline personality and trait anger and the covariance between them was decomposed into sources of A, D, and E. In this model the first variable loads only on the first factor and the second variable loads on the first two factors. Constraining the contributions of the latent factors of A or D at zero provides a test of whether these factors significantly contribute to the total variance in the traits. The significance of the genetic and environmental covariance structure was tested by constraining subsequent pathways ( $a_{21}$ ,  $d_{21}$ , and  $e_{21}$  in Figure 1) in the model at zero.

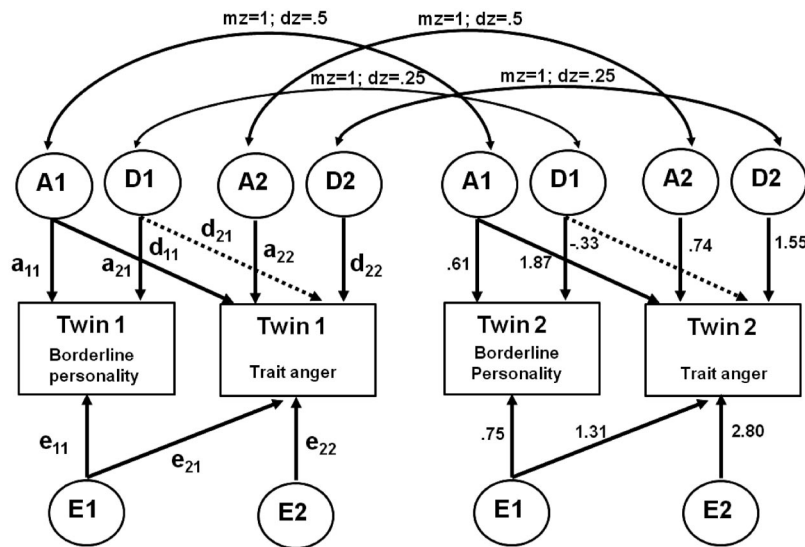


Figure 1. Bivariate genetic model; A1 and A2 = additive genetic factors; D1 and D2 = dominant genetic factors; E1 and E2 = unique environmental factors; a = factor loading of A; d = factor loading of D; e = factor loading of E. All latent A, D, and E factors have unit variance. For clarity reasons the nontwin sibling is not drawn.



All analyses were conducted using structural equation modeling in MX (Neale, Boker, Xie, & Maes, 2006). Testing of submodels was done by means of likelihood ratio tests, by subtracting the negative log-likelihood (-2LL) for the more general model from the -2LL of the more restricted model. This gives a  $\chi^2$  test with the degrees of freedom (*df*) equal to the difference in the number of estimated parameters in the two models. A significant  $\chi^2$  ( $p < .05$ ) indicates that the constrained model is significantly worse than the previous model and is therefore rejected. As a result, the previous model is kept as the most parsimonious model, to which a new model can be compared. In line with previous publication based on these data, a square root data transformation was performed for the borderline personality data but not for the trait anger data. To make sure this approach did not influence our results we reran all analyses with transformations for both measures but this did not change the results.

### Results

#### Tests of Fixed Effects on the Means and Variances

The borderline personality and the trait anger scores were not significantly different for twins and siblings,  $\chi^2_{(1)} = 0.008, p = .929$  for borderline and  $\chi^2_{(1)} = 0.053, p = .818$  for anger, and were dependent on age (all  $p < .01$ ). Both age regression coefficients were negative indicating that the borderline and anger scores decrease with age. A sex effect was only significant for borderline personality trait,  $\chi^2_{(1)} = 6.349, p = .012$ . Women had significantly higher borderline personality scores than men. In subsequent analyses the significant effects of sex and age were retained in the means model. Standard deviations were equal in males and females for both measures,  $\chi^2_{(1)} = 0.072, p = .788$  for borderline personality and  $\chi^2_{(1)} = 2.092, p = .148$  for trait anger.

#### Correlation Structure

Table 2 shows the twin and sibling correlations from the saturated model (upper part) and the correlations from the most constrained model (lower part). Table 3 gives the results of the tests performed on the correlation structure. The phenotypic correlation between borderline personality and trait anger scores did not differ for men and women and was estimated at .52 (Model 1). Correlations were similar for DZ twins and siblings for males and

females for both variables (Model 2). For both variables, the correlations were equal for DZ males and females and same sex siblings and for MZ males and females suggesting that the heritability is the same for men and women (Model 3). Additionally, the DZ and sibling same sex correlations were equal to the DZ and sibling opposite sex correlations indicating that the same genes influence the borderline personality and trait anger in men and women (Model 4).

#### Genetic Model

Based on the correlation structure, which does not provide evidence for the influence of C, and the results from prior studies (Distel et al., 2009; Torgersen et al., 2008; Kendler et al., 2008), we fitted an ADE model to the data. Genetic model-fitting results are summarized in Table 4. Removal of the dominant genetic effects (Model 1) resulted in a significant worsening of the goodness of fit ( $p = .027$ ). Dropping path  $d_{21}$  from the model did not result in a significant deterioration in model fit (Model 2;  $p = .237$ ), but dropping the path  $a_{21}$  (Model 3;  $p < .001$ ) and the path  $e_{21}$  (Model 4;  $p < .001$ ) did result in a significant decrease in model fit. This means that there are additive genetic and unique environmental factors that contribute to the covariance between the traits. Table 5 shows the estimates A, D, and E on variance in borderline personality and trait anger, the additive genetic and environmental correlations and the percentage of the phenotypic correlation explained by A and E. Figure 1 is a graphical representation of the bivariate model and gives the path coefficients from the best fitting model in the right part of the graph. The total variance in borderline personality can be written as  $(a_{11}^2) + (d_{11}^2) + (e_{11}^2)$ . The broad-sense heritability of borderline personality [calculated as  $(a_{11}^2) + (d_{11}^2)/(a_{11}^2) + (d_{11}^2) + (e_{11}^2)$ ] was estimated at 46%. The influence of E on individual differences in borderline personality [calculated as  $(e_{11}^2)/(a_{11}^2) + (d_{11}^2) + (e_{11}^2)$ ] was estimated at 54%. Total variance in trait anger can be written as  $(a_{21}^2) + (a_{22}^2) + (d_{21}^2) + (d_{22}^2) + (e_{21}^2) + (e_{22}^2)$ . The broad-sense heritability of trait anger [calculated as  $(a_{21}^2) + (a_{22}^2) + (d_{21}^2) + (d_{22}^2)/(a_{21}^2) + (a_{22}^2) + (d_{21}^2) + (d_{22}^2) + (e_{21}^2) + (e_{22}^2)$ ] was estimated at 40%. The influence of E on trait anger [calculated as  $(e_{21}^2) + (e_{22}^2)/(a_{21}^2) + (a_{22}^2) + (d_{21}^2) + (d_{22}^2) + (e_{21}^2) + (e_{22}^2)$ ] was estimated at 60%. The additive genetic and environmental covariance may be written as  $(a_{11} \times a_{21})$  and  $(e_{11} \times e_{21})$ , respectively. The dominant genetic covariance did not significantly explain covariance be-

Table 2  
Twin and Sibling Correlations for Trait Anger and Borderline Personality

	Twin correlation trait anger	Twin correlation borderline personality	Cross-twin cross-trait correlation
Monozygotic males twin pairs	.31	.46	.27
Dizygotic male twin pairs	.14	.23	.11
Monozygotic female twin pairs	.44	.46	.29
Dizygotic female twin pairs	.12	.15	.09
Dizygotic opposite sex twin pairs	.14	.20	.12
Brothers	.15	.14	.10
Sisters	.17	.24	.15
Brother-sister pairs	.11	.13	.08
All monozygotic twins	.41	.47	.29
All dizygotic twins/siblings	.14	.18	.12

Table 3  
Model Fit Results for the Saturated Bivariate Model

Model	Test	Versus	-2LL	df	$\chi^2$	$\Delta df$	p
0	Full model	—	56053.875	13855			
1	Phenotypic correlation males = Phenotypic correlation females	0	56053.885	13856	.01	1	.920
2	Twin correlations = sibling correlations	1	56059.194	13865	5.309	9	.807
3	Male correlations = female correlations	2	56068.141	13871	8.947	6	.177
4	<b>Same sex DZ/sibling correlations = opposite sex DZ/sibling correlations</b>	<b>3</b>	<b>56069.810</b>	<b>13874</b>	<b>1.669</b>	<b>3</b>	<b>.644</b>

Note. -2LL = -2 log likelihood; df = degrees of freedom; p = p-value; DZ = dizygotic. The best fitting model is printed in bold.

tween the traits. The additive genetic correlation ( $r_{Ga}$ ) [calculated as  $a_{11} \times a_{21} / (\sqrt{a_{11}^2} \times \sqrt{a_{21}^2 + a_{22}^2})$ ] was estimated at .93 and the environmental correlation [calculated as  $e_{11} \times e_{21} / (\sqrt{e_{11}^2} \times \sqrt{e_{21}^2 + e_{22}^2})$ ] at .42. The percentage of the phenotypic correlation explained by A may be calculated as  $\sqrt{h_{bpd}^2} \times r_G \times \sqrt{h_{anger}^2}$  divided by the phenotypic correlation. Likewise, the percentage of the phenotypic correlation explained by E may be calculated as  $\sqrt{e_{bpd}^2} \times r_E \times \sqrt{e_{anger}^2}$  divided by the phenotypic correlation. Based on these calculations the phenotypic correlation can be explained for 54% by A and 46% by E.

**Replication Using the 22-Item PAI-BOR Scale**

We repeated all analyses after deleting the two PAI-BOR items that directly tapped anger from the total PAI-BOR score for each participant. The same pattern of results was obtained. Specifically, the phenotypic correlation was estimated at 0.50 as compared with .52 in the original analyses. The correlation pattern was similar to the original analyses. The MZ twin correlations and DZ twin/sib correlations were estimated at 0.41 and 0.14 for trait anger, and at 0.46 and 0.18 for borderline personality, respectively. The cross correlation was estimated at 0.28 for MZ twins and at 0.11 for DZ twins and siblings. The heritability estimated found in the genetic analyses were equal to those found in the original analyses. Only the genetic correlation decreased from 0.93 to .89 and the unique environmental decreased from 0.42 to 0.40.

**Discussion**

Both trait anger and borderline personality are influenced by additive genetic, dominant genetic, and unique environmental factors. These findings are in line with previous studies reporting heritability estimates for BPD, borderline personality scores, and

trait anger scores. Previous research showed that there is a moderate to high phenotypic correlation between trait anger and BPD (Dolan, Anderson, & Deakin, 2001; Morse et al., 2009; Newhill, Eack, & Mulvey, 2009). The basis of the overlap however remained unclear in these previous studies. In the present study, structural equation modeling was applied to disentangle the relative influence of the genes and the environment on the covariance between the trait of borderline personality and trait anger. Significant correlations between the latent additive genetic and unique environmental factors that influence the two traits were found. Results showed that the phenotypic association ( $r = .52$ ) could be explained by additive genetic factors that are shared between the traits (54%) and by shared unique environmental influences (which also includes some measurement error; 46%). A similar level of correlation ( $r = .50$ ) was found even after the two PAI-BOR items that directly indexed anger were deleted from the total PAI-BOR score. This result suggests that the moderate correlation between anger and borderline personality is not solely due to shared item content.

Shared genetic risk is thus one of the possible explanations for the association between trait anger and borderline personality. As mentioned earlier, one possible shared set of mechanisms that appear to characterize both those high in trait anger and those with significant borderline personality features is a cognitive style that includes selective attention to hostile social cues, a tendency to interpret the actions of others as potentially hostile or aggressive, and a propensity to ruminate over past transgressions by others or anger-provoking experiences in general (Owen, 2011). Attentional biases observed in BPD may help explain the tendency for those with this disorder to experience chronic negative affect that is interrupted only by intense episodes of fear, anxiety, or hostility (Carpenter, Bagby-Stone, & Trull, 2011). Whether the intense

Table 4  
Genetic Model Fitting Results for Borderline Personality and Trait Anger

Model	Test	-2LL	df	$\chi^2$	$\Delta df$	p
0. ADE model	—	52603.736	12994	—	—	—
1. AE model	1 vs. 0	52612.911	12997	9.175	3	.027
<b>2. Drop path d21</b>	<b>2 vs. 0</b>	<b>52605.132</b>	<b>12995</b>	<b>1.396</b>	<b>1</b>	<b>.237</b>
3. Drop path a21	3 vs. 2	52813.758	12996	208.626	1	<.001
4. Drop path e21	4 vs. 2	52915.985	12996	310.853	1	<.001

Note. v = versus; -2LL = -2 log likelihood; df = degrees of freedom; p = p-value; A = additive genetic factors; D = nonadditive genetic factors (dominance); E = unique environmental factors. The best fitting model is printed in bold.

Table 5  
*Estimates of the Contribution of the Additive and Dominant Genetic Factor and the Unique Environmental Factor to Variance in Borderline Personality Trait and Trait Anger, the Phenotypic Correlation, the Genetic Correlation, the Environmental Correlation and the Percentage of Covariance Between Borderline Personality and Trait Anger Explained by A and E*

	Borderline personality	Trait anger
Additive genetic factor (A)	.36	.25
Dominant genetic factor (D)	.10	.15
Unique environmental factor (E)	.54	.60
Phenotypic correlation ( $r_P$ )	.52	
Genetic correlation	.93	
Environmental correlation	.42	
% $r_P$ explained by A	54%	
% $r_P$ explained by E	46%	

negative affect is experienced and expressed as anger or as fear, for example, may depend on nature of the situation that arouses the negative affect; anger is more likely to be aroused if approach tendencies (goals) are blocked whereas fear or anxiety arise from avoidance motivation (i.e., experience of threat; Carver & Harmon-Jones, 2009). It is also worth noting that deficits in the recruitment of effortful control resources (e.g., Rothbart, 1989) in hostility-related contexts may play a role in the frequency, duration, and intensity of anger expression (Wilkowski & Robinson, 2010; Wilkowski & Robinson, 2010). Such a cognitive set characterized by these attentional biases, biased appraisals, ruminative tendencies, and deficits in effortful control may contribute to BPD features such as conflicted interpersonal relationships, impulsive behavior, paranoid ideation, and even self-harm behaviors, in addition to intense negative affects.

These cognitive processes described above, are activated by environmental circumstances that are likely unique to the individual (i.e., nonshared environmental influence [e.g., rejection]). This is in line with the moderate degree of correlation between unique environmental influences on trait anger and on borderline personality found in this study. As there are no known specific environmental events that are unique to the development of BPD or trait anger, another possibility to consider is the concept of an “invalidating environment,” which is a major component of Linehan’s biopsychosocial model of BPD (Linehan, 1993). Briefly, Linehan’s model highlights the transactional process between emotional hypersensitivity and the experience of an invalidating environment, a process starting in childhood, which in turn leads to major features of BPD. In her theory, an invalidating environment is one that communicates to the individual that her or his emotional/internal experience or behavior is inappropriate or wrong. Thus, it is not a specific environmental event per se, but rather an encounter with others in the person’s unique environment context that contributes to the “invalidating” experience. Therefore, invalidation may take the form of abuse, neglect, or excessive criticism for example. Further, it is important to note that not all (e.g., siblings) are equally vulnerable to invalidation. Based on the standard quantitative genetic model (Falconer & Mackay, 1996) we know that siblings within the same family have correlated, but

different genotypes. Linehan proposes that those who are temperamentally emotionally sensitive are most vulnerable to the effects of invalidation. It is important Linehan (1993) notes that the consequences of invalidation include increased emotional arousal, and negative affects such as anger. Therefore, we speculate that this experience of an invalidating environment may be at least partially responsible for the finding of a correlation between environmental influences on trait anger and on borderline personality scores. More research will be necessary to directly test this proposal.

When interpreting the outcomes of this study, some limitations should be kept in mind. First, as we noted above, the PAI-BOR questionnaire includes two items (items 10 and 18) that explicitly deal with anger. Although trait anger and anger as part of BPD are two related but different concepts, this might have influenced the results. Therefore, we reran the analyses excluding the two anger related items from the PAI-BOR. The genetic architecture however did not change, nor did the general pattern of results. Second, nonresponse may limit the validity of questionnaire studies when nonresponse is associated with the traits under study. Distel et al. (2007) suggest that nonresponse may be higher among subjects with more BPD features because the participating members of less cooperative families showed somewhat higher scores on the PAI-BOR scale compared to member of highly cooperative families. However, the difference in borderline personality scores between less and highly cooperative families was quite small so the practical importance of this difference should not be overestimated. For trait anger, no significant association with nonresponse was found (Distel et al., 2007). Third, self-report questionnaires were used to assess borderline personality and trait anger. Although interview data and questionnaire data for BPD are highly correlated (Kurtz & Morey, 2001), results should be generalized to clinical populations and to a BPD diagnosis specifically with caution. Finally, the present study did not take possible gene–environment (GE) correlation or interaction into account (Livesley, 2008). If GE interaction plays an important role, its effects would be included in the “E” component of the model, and thus the role of genetics might be larger than indicated by the current results. If GE correlation (the nonrandom distribution of genotypes across environments) is present, part of the genetic influences derive from the effect of GE correlation (Rutter, 2007). GE correlation was suggested for borderline personality and certain life events (Distel et al., 2011). Future studies focusing on identifying the genes and the environmental factors that influence both trait anger and borderline personality will help us better understand the covariance of these traits.

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