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Familial factors and the risk of borderline personality pathology: genetic and environmental transmission

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Abstract

Background. Parental characteristics and practices predict borderline personality disorder (BPD) symptoms in children. However, it is difficult to disentangle whether these effects are genetically or environmentally mediated. The present study examines the contributions of genetic and environmental influences by comparing the effects of familial risk factors (i.e. parental psychopathology and borderline traits, maladaptive parenting, marital discord) on child BPD traits in genetically related (biological) and non-related (adoptive) families.

Methods. Data are from 409 adoptive and 208 biological families who participated in the Siblings Interaction and Behavior Study (SIBS) and 580 twin families the Minnesota Twin Family Study (MTFS). Parent characteristics and practices included parental psychopathology (measured via structured clinical interviews), parental BPD traits, parenting behaviors, and marital discord. A series of multi-level regression models were estimated to examine the relationship of familial risk factors to child BPD traits and to test whether children's adoptive status moderated the association.

Results. Symptom counts of parents' conduct disorder, adult antisocial behavior, nicotine, alcohol, and illicit drug dependence, and paternal BPD traits substantially predicted child BPD traits only in biological offspring, implying genetic transmission. Maternal BPD traits and both maternal and paternal conflict, lack of regard, and lack of involvement predicted offspring BPD traits regardless of the adoptive status, implying environmental transmission.

Conclusions. Parental externalizing psychopathology and father's BPD traits contribute genetic risk for offspring BPD traits, but mothers' BPD traits and parents' poor parenting constitute environmental risks for the development of these offspring traits.

Borderline personality disorder (BPD) is a debilitating mental illness with a lifetime prevalence of 2 to 6% in the general population (Grant *et al.*, 2008; Tomko *et al.*, 2014) and about 15 to 27% in clinical populations (Korzekwa *et al.*, 2008). BPD is characterized by emotional intensity and instability, identity disturbance, impulsive and self-destructive behaviors, and chaotic interpersonal relationships (American Psychiatric Association, 2013). BPD is a major public health concern as it is associated with high distress, functional impairments, and utilization of mental health resources both cross-sectionally (Bender *et al.*, 2001) and longitudinally (Zanarini *et al.*, 2004; Bagge *et al.*, 2005; Winograd *et al.*, 2008). As such, there is a clear need to understand the factors contributing to the etiology and maintenance of this disorder (Brune, 2016).

The influences of parental psychopathology, parent–child interactions, and family environment have been prominent in both theoretical frameworks of BPD (Kernberg, 2004; Fonagy and Luyten, 2009) and in empirical research (Paris, 2000; Zanarini, 2000; Gratz *et al.*, 2011; Belsky *et al.*, 2012). Several family studies document the aggregation of depression, antisocial personality disorder, and substance-use disorders – as well as BPD proper – in family members of BPD probands (Soloff and Millward, 1983; Zanarini *et al.*, 1998; White *et al.*, 2003; Zanarini *et al.*, 2009). One of the most influential theories to date, Linehan's biopsychosocial theory of BPD, makes a strong emphasis on the role of family environment and environmental adversity in the development of BPD (Linehan, 1987, 1993). Several retrospective and prospective studies document the role of family factors such as parenting styles, divorce, and marital discord (Frank and Paris, 1981; Zweig and Paris, 1991; Bandelow *et al.*, 2005; Winsper *et al.*, 2012). For instance, individuals with BPD recall lower parental bonding as well as higher parental control (Zweig and Paris, 1991). Parent-reported childhood neglect predicted increased BPD severity 10 years later during adolescence and early adulthood (Johnson *et al.*, 2000). Furthermore, parents with BPD seem to engage in negative parenting strategies that in turn are associated with an increased likelihood of BPD in their offspring (Newman *et al.*, 2007; Stepp *et al.*, 2012; Zalewski *et al.*, 2014).

Interpretation of these studies, however, is far from straightforward. A series of recent studies document moderate (≈ 40%) genetic contributions to BPD traits and symptoms (Distel

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et al., 2008; Reichborn-Kjennerud *et al.*, 2013; Amad *et al.*, 2014; Reichborn-Kjennerud *et al.*, 2015). 'Environmental' factors such as marital relationship (Spotts *et al.*, 2004), divorce (Jocklin *et al.*, 1996), and parenting styles (Kendler *et al.*, 2011) are also heritable (see Kendler and Baker, 2007 for a review). It is possible that patterns of influence in BPD generally attributed to the familial environment are, in fact, at least partly genetically mediated. Putative environmental factors may be indirectly related to offspring BPD traits through the mechanism of passive gene–environment correlation (r_{GE}; Plomin *et al.*, 1977, Scarr and McCartney, 1983). Passive r_{GE} occurs when parents create an environment that correlates with their genotype, while also passing down their genotype to their offspring. Therefore, the association between environmental factors and child outcomes is spurious and could be a function of shared genetics. In the current context, heritable factors may increase the likelihood of parental psychopathology, marital conflict and maladaptive parenting among the parents, and BPD features in the offspring.

Unfortunately, data solely from families reared together cannot help to accurately partition genetic and environmental influences in the presence of gene–environment correlation or direct effects, as in this context, parents provide both the genetic risk and the social environment for their offspring. Classical twin studies also cannot account for the effects of passive r_{GE} and can incorrectly model them as shared environmental effects (Carpenter *et al.*, 2013). An effective method of disentangling direct environmental from passive r_{GE} effects is comparing the patterns of association in biological and adopted children. Because adoptive children do not inherit their genotype and environment from the same source, the effect of passive r_{GE} is eliminated. If familial risk factors are associated with BPD traits in biological and adopted offspring equally, it would imply a direct environmental influence. On the contrary, if the risk factors are only related to BPD traits in biological children, it would imply genetic transmission. To our knowledge, the effect of putative environmental risk factors and parental psychopathology on the development of offspring BPD features has not been examined through an adoptive design.

The current study aimed to examine the vertical transmission of parent psychopathology, parenting practices, and family discord on offspring BPD traits, while accounting for the relative contribution of passive r_{GE}. Using a large sample of biological and adoptive families recruited from the community, we aimed to estimate the relative contribution of genetic and direct environmental effects. Among the familial risk factors, we included parent psychopathology, parenting practices, and marital discord. Maternal and paternal psychopathology included BPD traits, adult antisocial behavior, history of conduct disorder, nicotine, alcohol and illicit substance dependence, and major depressive disorder. Notably, although parental psychopathology is not strictly a measure of 'environment', the adoptive design still allowed us to broadly examine genetic and environmental effects. In addition to maladaptive parental practices, we examined the effects of marital discord on offspring BPD traits.

Hypotheses

Rather than having a single directional hypothesis, we had the following competing expectations for each familial risk factor. If only environmental transmission is present, then parental risk factors should predict offspring BPD traits equally in adoptive and biological offspring. If only genetic transmission is present, one would expect an effect in biological offspring, a zero or negligible effect in adoptive offspring (as well as a significant interaction between parental risk factors and offspring adoptive status if both family types are modeled together). Finally, if both environmental and genetic transmissions are operating, then there should be effects in both biological and adoptive offspring, with the effect being larger in biological offspring (i.e. a significant interaction between parental risk factors and adoptive status).

Method

Sample

The current analyses used data from two large studies. The Siblings Interaction and Behavior Study (SIBS) (McGue, Keyes *et al.*, 2007) at the University of Minnesota consisted of 409 adoptive and 208 biological families [$M_{age\ offspring} = 14.93$ (S.D. = 1.93); 55% female; 56% Caucasian, 39% Asian, and 5% other], recruited between 1998 and 2005. Families were eligible if they had two offspring between the ages of 11 to 21 who did not have a physical or mental impairment that could hinder the assessment. Adoptive families were ascertained from infant placements made by the three largest, private adoption agencies in Minnesota. Non-adoptive families were ascertained through Minnesota state birth records and selected to have a pair of siblings of comparable age and gender to the adoptive sibling pairs. For both adoptive and non-adoptive families, the siblings had to be no more than 5 years apart in age. For adoptive families, the adopted offspring had to have been placed prior to age 2 years. Mean age of permanent placement in the sample was 4 months. Families who had one adopted and one biological child were also eligible. Of the adoptive families, there were 124 'mixed' family structures, with one adopted child, and one child biologically related to at least one parent. Participation rate was high for adoptive (63.2%) and non-adoptive (57.3%) families. Previous work (McGue *et al.*, 2007) documented few differences between participating and recruited but non-participating families, or between the study families and the general community.

The Minnesota Twin Family Study (MTFS) consisted of 580 families consisting of parents and twin pairs [$M_{age\ offspring} = 14.90$ (S.D. = 0.60); 93.1% Caucasian]. Only female twins were included, because personality data was not available for male twins at age 14. MTFS is an ongoing population-based, longitudinal study of twins and their families (Iacono *et al.*, 1999; Keyes *et al.*, 2009). Birth records and public databases were used to locate more than 90% of families that included a twin birth in the state of Minnesota from 1975 to 1984 and from 1988 to 1994. Eligible twins and their families (a) were living within a 1-day drive of Minneapolis with at least one biological parent, and (b) had no mental or physical handicap precluding participation. Parental psychopathology was assessed at intake (when twins were aged ≈11) and again 6 years later, allowing us to obtain a lifetime index of parental psychopathology. BPD traits were first assessed at age 14 among female twins; parenting and marital satisfaction were measured concurrently. We used the age-14 assessment for the current analyses and included all families where at least one offspring had BPD trait data. Previous work (Johnson *et al.*, 2002) reported few differences in personality between twins and singletons in the community.

Measures

Psychopathology

The Structured Clinical Interview for DSM-III-R (Spitzer *et al.*, 1987), updated to include the DSM-IV criteria, was administered to both parents to assess symptoms of adult antisocial behavior, history of conduct disorder, and major depressive disorder. Higher scores reflect

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a higher number of symptoms endorsed. Parental symptom counts of nicotine, alcohol and illicit substance dependence (cannabis; opiates; amphetamine; sedatives; hallucinogens; cocaine; phencyclidine; inhalants) were measured using the substance use supplement of the Composite International Diagnostic Interview (Robins *et al.*, 1987). The inter-rater reliability for all diagnostic procedures was higher than $\kappa = 0.89$ (Iacono *et al.*, 1999; McGue *et al.*, 2007). For parents and offspring aged 16 and older, BPD traits were assessed using the Minnesota Borderline Personality Scale (Bornovalova *et al.*, 2011), a 19-item, Likert-type, four-point self-report instrument derived from the Multidimensional Personality Questionnaire (Tellegen, 1982, 2003). For offspring younger than 16, BPD traits were assessed using the Personality Booklet Youth-Abbreviated (PBYA, developed specifically for the MTFS; e.g. Matteson *et al.*, 2013). The PBYA contains 17 of the 19 items on the MBPD. To make the MBPD and PBYA compatible, the PBYA score was prorated to a score compatible with MBPD (range 19–76) by using the proportion of maximum possible scaling method (POMP; Cohen *et al.*, 1999). Higher scores are indicative of higher borderline personality pathology. The MBPD has excellent psychometric properties and has been validated on both adult and mid- to older adolescent samples (Rojas *et al.*, 2014; Rojas *et al.*, 2015)[†1]. In the current sample, the MBPD the PBYA showed good internal consistency (MBPD, $\alpha = 0.82$; PBYA, $\alpha = 0.84$). Notably, the correlations between the mother's and father's psychopathology were low (all $rs < 0.30$ between parents), and as such, parental psychopathology variables were kept separate in all analyses.

Family environment

The Parental Environment Questionnaire (Elkins *et al.*, 1997) is a 50-item self-report instrument that measures parent–child interactions. Each parent responded to the PEQ for each offspring, using a four-point scale to yield subscales of conflict, lack of involvement, and lack of regard (αs ranged from 0.78 to 0.87). Additionally, each parent's opinions on favoring punishment were also assessed using nine items on a child rearing questionnaire (αs ranged from 0.75 to 0.81). Higher scores on the PEQ and attitudes towards punishment reflected higher maladaptive parenting. As above, the correlations between the parents' styles were low (all $rs \leqslant 0.30$), except for conflict and punishment, which were higher (both $rs = 0.43$).

Marital discord was measured using a 34-item modified version of the Dyadic Adjustment Scale (Spanier, 1976), with two additional items concerning parents' attitudes towards child rearing. The DAS taps into marital satisfaction, and cohesion and marital consensus, and higher scores reflect high marital discord ($\alpha = 0.93$). Each parent rated the relationship separately and their scores were averaged together to form a single estimate for each couple ($rs > 0.60$ between parent reports), where higher scores reflected higher discord. Finally, mothers also reported separation/divorce status. In our sample, 87.8% of the mothers were currently married and 12.2% were divorced/separated from the biological father; the remaining either never married or widowed[2].

Data analyses

As shown in Table 1, there were significant differences between biological and adoptive parents. Adoptive parents were older, were more likely to finish college and/or post-graduate school, had higher income, and were more likely to be Caucasian than biological parents. Subsequently, we adjusted for parental age, family socioeconomic status, and parental race in all analyses, and all psychological measures were standardized. We also adjusted for offspring age, sex, and ethnicity, as sex and ethnicity showed substantial differences across adoptive *v.* biological groups, and previous work indicates sex as well as age and ethnicity effects on BPD traits (Shea *et al.*, 2009; Silberschmidt *et al.*, 2015).

Given dependence among data for siblings from the same family, we conducted our analyses using multilevel models with the *lme4* (version 1.1-21; Bates *et al.*, 2015), *optimx* (version 2018-7.10; Nash and Varadhan, 2011), and *simr* (version 1.0.5; Green and MacLeod, 2016) packages in *R* (version 3.5.2; R Core Team, 2018). All analyses were estimated as two-level random-intercept models with intercepts allowed to vary across families and residual variances allowed to differ across adoptive, biological sibling/DZ, and MZ groups. Each parental risk factor was examined separately. Across models, offspring-level covariates and predictors [age, sex, ethnicity, adoptive status, parenting practices (conflict, lack of regard, lack of involvement)] were included at level-1. Parent- and family-level covariates and predictors [parent age and ethnicity, socioeconomic status, parent psychopathology, parenting practices (attitude toward punishment), marital discord, divorce] were included at level-2.

We fit a series of two-level random-intercept models to test three key questions: (1) Is there a practically significant effect of familial variables on offspring BPD traits within adoptive families? (2) Is there a practically significant effect within biological families? and (3) Is the effect within biological families practically significantly greater than the effect within adoptive families? Evidence for environmental transmission is implied by the presence of both (1) and (2), whereas evidence for genetic transmission is implied by (2) and (3). Note that a combination of genetic and environmental transmission is inferred when (1), (2), and (3) are all present. Questions (1) and (2) were examined by estimating models separately in the adoptive and biological offspring samples. Each model included the covariates (parent age and ethnicity, family socioeconomic status, offspring gender, age, and ethnicity) and a familial risk factor (parental psychopathology or family environment) as predictors, with offspring BPD traits as the outcome. Question (3) was tested using the combined full sample with moderated regression analyses as a formal test of slope differences across biological and adoptive samples. Each model included the above covariates and a familial risk factor as predictors. Question 3 also included adoptive status and the interaction between adoptive status and the familial risk factor. A significant (i.e. one with a non-negligible effect size where the confidence interval does not include zero) moderation effect by adoptive status indicates the potential impact of the familial risk factor for BPD traits differs across biological and adoptive offspring, thereby supporting an interpretation of genetic transmission. Notably, instead of solely focusing on statistical significance, we interpret the broad pattern of effects and the magnitudes of effect sizes. To this end, all effect sizes were standardized and can be interpreted using empirical effect size benchmarks identified by Gignac and Szodorai (2016) (Cohen's *d*: 0.2, 0.5, 0.8; Pearson's *r*: 0.1, 0.2, 0.3 for small, medium, and large, respectively corresponding to the quartiles for effect sizes observed in empirical individual differences research). Standardized regression coefficients (β) were interpreted in the *r* metric.

Results

Differences between families in parent and offspring psychopathology are displayed in Table 2. Adoptive offspring reported slightly higher BPD traits. Biological and adoptive families showed small but significant differences on most other factors, including higher

†The notes appear after the main text.

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Table 1. Demographic characteristics in adoptive and biological families

	Adoptive	Biological	
	M (s.d.)/%	*M* (s.d.)/%	*d*/*diff.* % (95% *CI*)
Mothers			
Age	48.06 (3.39)	43.70 (4.79)	−0.43 (−0.52 to −0.34)
% College graduate	60.61%	35.43%	−0.52 (−0.61 to −0.43) −25.01% (−29.25% to −20.63%)
% Caucasian	98.54%	96.41%	−0.12 (−0.21 to −0.03) −2.13% (−3.33% to −0.68%)
Fathers			
Age	49.27 (3.62)	46.32 (4.66)	−0.30 (−0.39 to −0.21)
% College graduate	64.74%	37.84%	−0.55 (−0.64, −0.46) −26.90% (−31.10% to −22.53%)
% Caucasian	97.78%	95.34%	−0.12 (−0.21 to −0.03) −2.45% (−3.86% to −0.77%)
% Family income >$80 000	58.06%	32.18%	−0.49 (−0.58 to −0.40) −25.88% (−30.57% to −21.04%)
Offspring			
Age	14.94 (1.95)	14.90 (1.17)	−0.03 (−0.12 to 0.06)
% Female	55.62%	85.67%	0.75 (0.66 to 0.84) 30.05% (25.97% to 34.12%)
% Caucasian	22.53%	94.95%	2.42 (2.31 to 2.54) 72.43% (68.89% to 75.60%)

For categorical differences, both *d* (Cohen's *d*) and *diff.* % (difference in percentage) were calculated. Negative *d* or *diff.* % values indicate higher means for adoptive offspring. Among parents, data were available on at least one parental variable for 2356 moms and 2129 dads.

levels of maternal and paternal externalizing psychopathology and BPD traits. Adoptive parents reported a significantly higher parent–child conflict, but were less likely to favor punishment. Biological and adoptive families did not differ on the level of marital discord, but the former were more likely to be divorced.

Table 3 summarizes the association between familial risk factors and offspring BPD traits in adoptive and biological offspring separately, as well as for interaction effects that test for slope differences across family types[3]. Evidence of a genetic effect required a practically significant relationship ($\beta \geq .10$) between offspring BPD traits and the familial risk factor for biological families and a practically significant interaction effect. This pattern was observed for both parents' psychopathology for conduct disorder, adult antisocial behavior, and nicotine, alcohol, and illicit substance dependence, with small to moderate positive effects in biological families and similar sized interaction effects (β_{ado} ranged −0.08 to +0.03, β_{bio} ranged 0.07 to 0.16, β_{int} ranged 0.12 to 0.25; see Fig. 1 for visual representations[4]). The confidence interval for paternal illicit substance dependence included zero but the pattern and magnitude of effects were consistent with other externalizing psychopathology ($\beta_{ado} = 0.01$, $\beta_{bio} = 0.11$, $\beta_{int} = 0.12$) and coefficients were not significantly different from coefficients for maternal illicit substance dependence. For major depression, maternal and paternal effects were weak and similar, with limited to little support evident for the transmission of either environmental or genetic risk (for maternal and paternal effects, respectively: $\beta_{ado} = 0.08, 0.01$, $\beta_{bio} = 0.10, 0.07$, $\beta_{int} = 0.03, 0.04$). Paternal BPD traits displayed the genetic effect pattern ($\beta_{ado} = 0.04$, $\beta_{bio} = 0.19$, $\beta_{int} = 0.15$), but maternal BPD traits showed the environmental effect pattern, with consistent small relationships with offspring BPD across family types ($\beta_{ado} = 0.15$, $\beta_{bio} = 0.15$, $\beta_{int} = -0.01$).

Evidence of an environmental effect required a practically significant relationship ($\beta \geq .10$) between offspring BPD traits and the familial risk factor for both adoptive and biological families. As noted above, this pattern was observed for maternal BPD traits. It was also observed for both maternal parenting practices (conflict, lack of regard, lack of involvement; β_{ado} ranged 0.16 to 0.28, β_{bio} ranged 0.19 to 0.24, β_{int} ranged −0.01 to 0.00), and paternal parenting practices (conflict, lack of regard, lack of involvement; β_{ado} ranged 0.11 to 0.18, β_{bio} ranged 0.09 to 0.21, β_{int} ranged −0.03 to +0.02). Parental attitudes toward punishment and marital discord had limited support evident for either environmental or genetic transmission (for maternal punishment, paternal punishment, and marital discord respectively: $\beta_{ado} = 0.09, 0.08, -0.01$; $\beta_{bio} = 0.07, 0.10, 0.10$; $\beta_{int} = 0.01, 0.05, 0.10$).

Evidence for both a genetic and an environmental effect required practically significant effects for both adoptive and biological families, with a larger effect in biological families (i.e. a significant interaction). No such pattern was observed.

In summary, the small to medium effects of parental externalizing disorders (maternal and paternal conduct, antisocial, and substance use disorders, paternal BPD) appeared to reflect primarily genetic effects, whereas the small to medium effects of maternal BPD and maternal and paternal parenting practices appeared to reflect primarily environmental effects.[5]

Discussion

The goal of the current investigation was to determine if the vertical transmission of parent psychopathology on offspring BPD traits is explained by genetic factors, direct environmental influence of parenting and home environment, or both. To our

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Table 2. Summary statistics of parent and offspring psychopathology

	Adoptive	Biological	
	M (s.d.)/%	*M* (s.d.)/%	*d*/*diff.* % [95% CI]
Offspring BPD Traits	41.73 (9.32)	40.96 (9.48)	−0.11 (−0.20 to −0.02)
Maternal psychopathology			
Conduct disorder	0.20 (0.59)	0.31 (0.70)	0.16 (0.07 to 0.25)
Adult antisocial behavior	0.67 (0.71)	0.90 (0.92)	0.27 (0.18 to 0.36)
Nicotine dependence	0.74 (1.35)	1.01 (1.6)	0.18 (0.09 to 0.27)
Alcohol dependence	0.19 (0.83)	0.32 (0.95)	0.15 (0.06 to 0.24)
Illicit substance dependence	0.12 (0.64)	0.31 (1.05)	0.21 (0.12 to 0.3)
Major depression	1.80 (2.66)	1.71 (2.76)	−0.03 (−0.12 to 0.06)
Borderline personality traits	31.63 (6.54)	33.40 (7.14)	0.26 (0.17 to 0.34)
Paternal psychopathology			
Conduct disorder	0.74 (1.12)	.95 (1.41)	0.16 (0.07 to 0.25)
Adult antisocial behavior	1.32 (0.91)	1.64 (1.18)	0.29 (0.20 to 0.38)
Nicotine dependence	1.16 (1.68)	1.30 (1.73)	0.08 (−0.01 to 0.17)
Alcohol dependence	0.59 (1.17)	1.00 (1.67)	0.27 (0.18 to 0.36)
Illicit substance dependence	0.25 (0.79)	0.62 (1.52)	0.28 (0.19 to 0.37)
Major depression	1.33 (2.37)	1.19 (2.35)	−0.06 (−0.15 to 0.03)
Borderline personality traits	32.35 (6.39)	33.9 (7.53)	0.22 (0.13 to 0.31)
Maternal parenting			
Conflict	52.00 (10.87)	49.32 (9.76)	−0.26 (−0.35 to −0.17)
Lack of regard	49.31 (9.67)	49.75 (10.21)	0.04 (−0.04 to 0.13)
Lack of involvement	50.46 (10.26)	49.71 (9.87)	−0.07 (−0.16 to 0.02)
Attitude toward punishment	48.43 (9.23)	50.35 (9.99)	0.21 (0.12 to 0.30)
Paternal parenting			
Conflict	51.30 (9.90)	49.27 (10.02)	−0.20 (−0.29 to −0.11)
Lack of regard	50.12 (10.33)	49.64 (9.92)	−0.05 (−0.14 to 0.04)
Lack of involvement	50.47 (9.91)	49.98 (10.18)	−0.05 (−0.14 to 0.04)
Attitude toward punishment	48.40 (9.56)	50.50 (10.07)	0.21 (0.12 to 0.30)
Marital discord	50.46 (9.70)	49.62 (9.89)	−0.09 (−0.17 to 0.00)
Divorce	6.40%	14.74%	0.25 (0.16 to 0.34) 8.31% (5.62 to 10.76%)

For categorical differences, both *d* (Cohen's *d*) and *diff.* % (difference in percentage) were calculated. Negative *d* or *diff.* % values indicate higher means for adoptive offspring. Symptom counts were used to index parental psychopathology, and the MBPD score (range 19–76) was used to index parental BPD traits. Parenting variables and marital discord were converted to *T* scores.

knowledge, this is the first study to test the association between familial risk factors and offspring BPD traits via the adoptive family design. Our results suggest that the route of transmission may vary based on the specific risk factors. Genetic transmission was found for externalizing psychopathology. Each parent's conduct disorder, adult antisocial behavior, and nicotine, alcohol, and illicit substance dependence, as well as paternal BPD traits substantially predicted offspring BPD traits in biological offspring only, and the difference in slopes between adoptive and biological offspring was substantial. The nicotine finding is also notable, as this is the first study to document the association between parental nicotine use and offspring BPD features and to specify a mechanism for this relationship. Findings are congruent with previous studies showing that BPD features have a strong genetic overlap with substance use frequency and dependence, and with antisocial behavior, both cross-sectionally (Kendler *et al.*, 2008; Hunt *et al.*, 2015) and longitudinally (Bornovalova *et al.*, 2013; Bornovalova *et al.*, 2018; Rosenstrom *et al.*, 2018). Indeed, family studies suggest there is high familial co-aggregation with disorders marked by behavioral disinhibition (White *et al.*, 2003). For instance, in the families of BPD probands, the risk for ASPD is 7–16% whereas the risks of alcohol and substance dependence are between 20% and 25% – rates much higher than in the general population (ASPD: 3.5%; alcohol dependence: 14%; drug dependence: 7.7%; Grant *et al.*, 2008). It is plausible that a common factor is inherited which acts as a general liability for related psychopathology. In the current context, a candidate common vulnerability factor in the transmission of parental externalizing

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Table 3. Familial risk factors and offspring BPD

	Mother			Father		
	Adoptive	Biological	Interaction	Adoptive	Biological	Interaction
Parent psychopathology						
Conduct disorder	0.03 (−0.05 to 0.11)	0.15 (0.07 to 0.23)	0.12 (0.01 to 0.23)	−0.04 (−0.13 to 0.04)	0.14 (0.06 to 0.22)	0.18 (0.05 to 0.30)
Adult antisocial behavior	−0.02 (−0.10 to 0.06)	0.14 (0.08 to 0.20)	0.15 (0.04 to 0.27)	−0.08 (−0.17 to 0.00)	0.16 (0.09 to 0.23)	0.25 (0.13 to 0.37)
Nicotine dependence	−0.04 (−0.12 to 0.04)	0.16 (0.10 to 0.22)	0.21 (0.10 to 0.32)	0.00 (−0.09 to 0.08)	0.15 (0.08 to 0.22)	0.14 (0.03 to 0.26)
Alcohol dependence	−0.06 (−0.14 to 0.02)	0.07 (0.01 to 0.13)	0.14 (0.03 to 0.26)	−0.05 (−0.14 to 0.04)	0.15 (0.08 to 0.22)	0.20 (0.07 to 0.34)
Illicit substance dependence	−0.06 (−0.13 to 0.02)	0.15 (0.09 to 0.22)	0.24 (0.11 to 0.38)	0.01 (−0.08 to 0.09)	0.11 (0.05 to 0.18)	0.12 (−0.04 to 0.27)
Major depressive disorder	0.08 (0.00 to 0.16)	0.10 (0.04 to 0.16)	0.03 (−0.08 to 0.13)	0.01 (−0.08 to 0.09)	0.07 (0.00 to 0.14)	0.04 (−0.07 to 0.15)
Borderline personality traits	0.15 (0.07 to 0.22)	0.15 (0.09 to 0.21)	−0.01 (−0.11 to 0.10)	0.04 (−0.05 to 0.12)	0.19 (0.12 to 0.26)	0.15 (0.03 to 0.27)
Parenting						
Conflict	0.28 (0.20 to 0.36)	0.24 (0.19 to 0.29)	−0.01 (−0.11 to 0.08)	0.18 (0.10 to 0.27)	0.21 (0.14 to 0.29)	0.02 (−0.09 to 0.14)
Lack of regard	0.16 (0.08 to 0.24)	0.19 (0.13 to 0.24)	0.00 (−0.10 to 0.11)	0.11 (0.02 to 0.20)	0.09 (0.01 to 0.18)	−0.02 (−0.14 to 0.10)
Lack of involvement	0.20 (0.12 to 0.28)	0.21 (0.16 to 0.27)	0.00 (−0.09 to 0.10)	0.13 (0.04 to 0.22)	0.10 (0.02 to 0.18)	−0.03 (−0.15 to 0.08)
Attitudes towards punishment	0.09 (0.01 to 0.18)	0.07 (0.01 to 0.13)	0.01 (−0.10 to 0.11)	0.08 (−0.01 to 0.17)	0.10 (0.03 to 0.17)	0.05 (−0.06 to 0.17)
Marital discord	−0.01 (−0.11 to 0.09)	0.10 (0.02 to 0.18)	0.10 (−0.03 to 0.23)	—	—	—

All columns present results from separate models with psychological measures standardized. Values are standardized regression coefficients (β) with 95% confidence intervals. All models controlled for offspring age, sex and ethnicity, parent ethnicity and age, and socioeconomic status (composite of income and both parents' education). Models with full sample also controlled for adoptive status. The variables *divorce* and *marital discord* each had a single value per family. Positive interaction coefficients indicate greater effect in the biological than adoptive offspring.

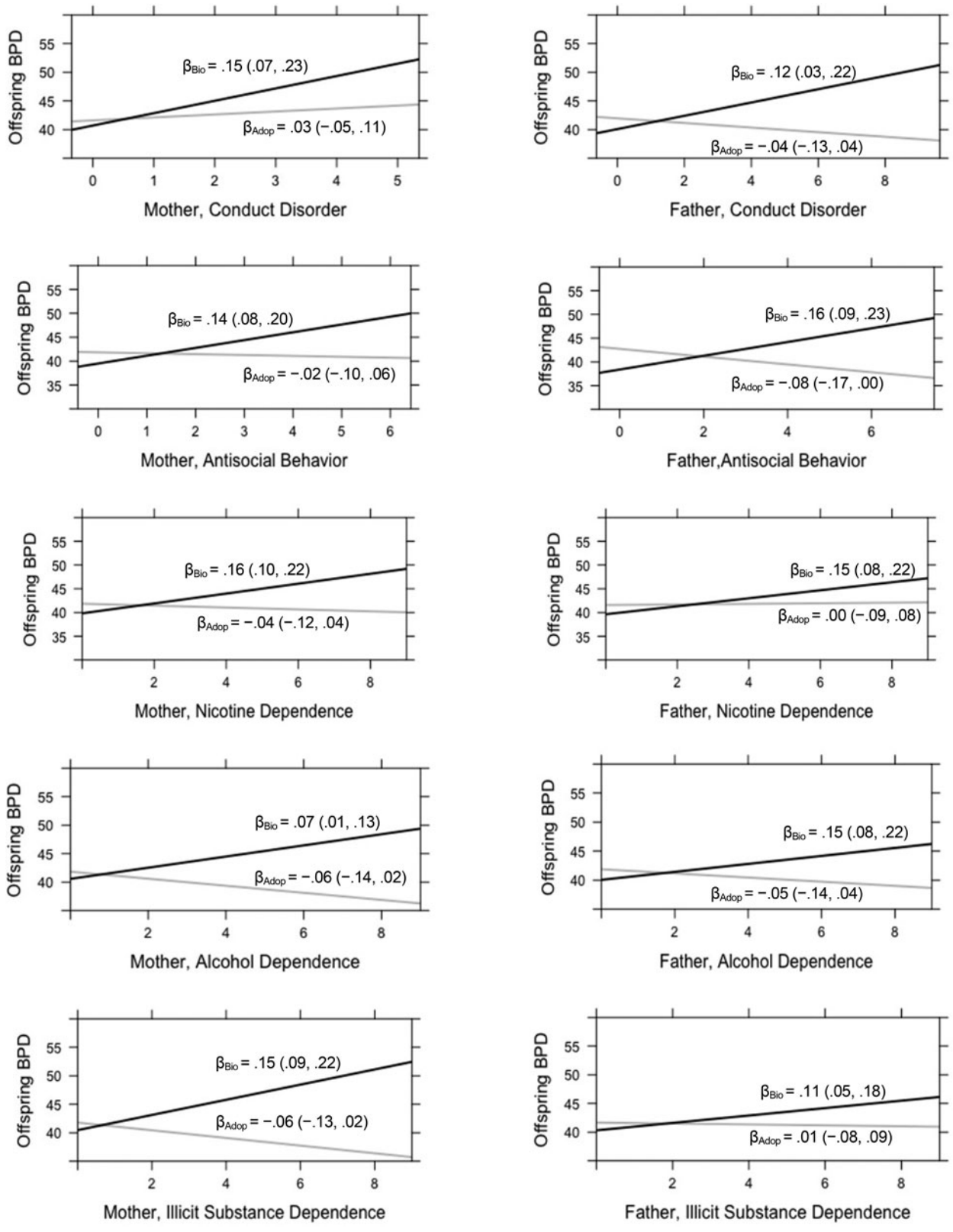

Fig. 1. Effects of familial risk factors on offspring BPD by adoptive status. Axis labels represent symptom counts for parental DSM-IV psychopathology and possible range (19–76) for parental BPD traits.

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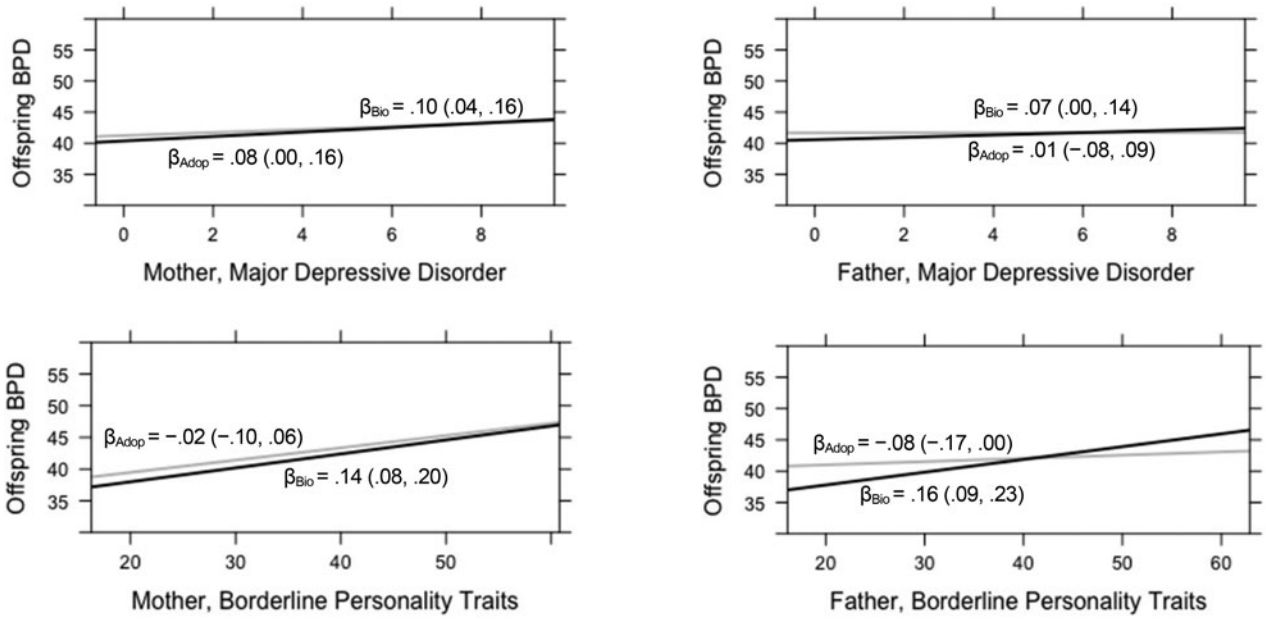

Fig. 1. Continued

variables to offspring BPD traits is behavioral disinhibition (Trull *et al.*, 2000), which is strongly heritable (Hicks *et al.*, 2013). Thus, it is likely that the genetically transmitted disposition toward behavioral disinhibition explains the association between parent externalizing and BPD traits in biological offspring.

Support for environmental transmission was found for maternal BPD and both maternal and paternal maladaptive parenting, including conflict, lack of involvement, and lack of regard. These variables predicted BPD traits with similar magnitudes in both biological and adopted offspring. This pattern of results is consistent with previous studies (Zweig and Paris, 1991; Golomb *et al.*, 1994). Previous studies document that mothers with BPD engage in negative parenting strategies (Stepp *et al.*, 2012), leading to subsequent poor psychosocial outcomes in offspring (Newman *et al.*, 2007). It is also consistent with several major etiological theories of BPD that highlight the role of familial environment (Weaver and Clum, 1993; Carr and Francis, 2009) and mother–offspring conflict (Crowell, Baucom *et al.*, 2013).

The current findings should be interpreted in the context of five limitations. First, although we controlled for measured differences in socioeconomic status between families, parents in adoptive families tended to be older and more educated and likely differed on other unmeasured variables as well. Second, we used a self-reported, dimensional/trait-based measure of BPD traits rather than a clinical interview-based diagnosis. While interviews are often assumed to be superior to questionnaire measures, there is limited evidence for the superiority or incremental validity of one method over the other, though each approach has unique strengths and weaknesses (Hopwood *et al.*, 2008). Additionally, we showed here that the MBPD has construct validity in participants as young as 11. Likewise, although the test-retest reliability of BPD in adolescents has been questioned (Biskin, 2015), this appears to be mainly a function of unreliability of dichotomized diagnoses. Indeed, several studies document that the test-retest reliability of BPD traits is high when measured via dimensional and/or trait-based indices (Chanen *et al.*, 2004). Yet ideally, the current study should be replicated using a multi-method approach (i.e. trait- and interview-based measures). Third, future studies should examine if the relationship between parental risk factors and offspring BPD traits varies by sex. Fourth, the data analyses were cross-sectional, and causal or directional statements require further assumptions (Rohrer, 2018). It is possible and indeed likely that offspring BPD traits evoke maternal–child conflict and lack of involvement, or that the influence is bidirectional (Stepp *et al.*, 2014). Fifth, the current study was able to arbitrate between environmental transmission *v.* passive r_{GE}. However, it was not able to detect or rule out evocative r_{GE} or $G \times E$. Evocative r_{GE} occurs when offspring characteristics evoke a response from the environment (e.g. offspring BPD traits lead parents to adopt harsher punishment practices); $G \times E$ occurs when offspring have a genetic susceptibility to environmental influences. These processes are plausible in the relationship between parenting and BPD traits from both a theoretical (e.g. Linehan, 1993) and empirical (Belsky *et al.*, 2012; Reinelt *et al.*, 2014; Stepp *et al.*, 2014) standpoint. Follow-up studies can disentangle these processes by moderating the genetic architecture of BPD traits by self- and parent-reports of parenting (Purcell, 2002).

Despite these potential limitations, several strengths of the study should be acknowledged. To our knowledge, this is the first paper that compared the transmission of BPD traits across biological and adoptive families and thus took the role of passive r_{GE} into account. Additionally, we used a large sample to test the hypotheses. Formal power analyses indicated we generally had sufficient power to detect even small effect sizes. Generally, BPD research involves retrospective self-reports on family environment and parenting practices. In this study, we measured current familial risk factors from parents themselves. Hence, we eliminated the influence of offspring's acute psychopathology and retrospective bias on reports of parent practices and home environment.

Findings of the current study contribute to the understanding of the etiology of BPD, which has implications for developing effective prevention and intervention strategies. Essentially, this study highlights the partial genetic nature of BPD while at the same time elucidating the role of parenting practices that are associated with BPD traits in both biological and adoptive

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offspring. Therefore, interventions targeting parenting practices could help prevent the expression of BPD traits in offspring.

Notes

[1] In previous work, we reported criterion-related validity of MBPD for adolescents aged 14 and older (e.g. Bornovalova et al. 2013, 2018). However, our sample included participants as young as 11, and it is of interest to know whether the MBPD is similarly valid for those between the ages of 11 and 14. We further anchored the assessment of BPD traits by constructing a nomological network of the MBPD in our overall sample, followed by the same analyses in younger versus older subsamples (<14 *v.* >14). In the overall sample, offspring MBPD correlated with clinician-ascertained symptom counts of offspring: ADHD ($r = 0.32$), ODD (0.38), CD (0.32), MDD (0.28), nicotine dependence symptoms (0.26), alcohol use disorder symptoms (0.15), and drug use disorder symptoms (0.14) (all $ps < 0.01$). Among those aged <14 ($N = 457$), MBPD correlated with ADHD ($r = 0.39$), ODD (0.43), CD (0.28), MDD (0.18), and nicotine dependence (0.13) (all $ps < 0.01$); very few adolescents reported alcohol, or drug use disorder symptoms before age 14. Among those aged >14 ($N = 1909$), MBPD correlated with: ADHD ($r = 0.30$), ODD (0.37), CD (0.33), MDD (0.30), nicotine dependence symptoms (0.28), alcohol use disorder symptoms (0.17), and drug use disorder symptoms (0.15) (all $ps < 0.01$). The pattern of correlations was similar between males and females. Thus, even among the younger participants, MBPD shows criterion-related validity and functions much like the BPD construct in the literature (e.g. Stepp et al. 2012).

[2] Formal power analyses indicated low power to detect the interaction between adoptive status and divorce. As such, this variable was not analyzed further for passive r_{GE}.

[3] Full models including covariates are included on https://osf.io/6c8jx/, along with power curves for the interaction term for all models. Formal power analyses indicated we generally had ~75% power and better to detect effects of 0.15–0.2, and about ~50–60% to detect effects of 0.1.

[4] Figures for all variables are available on OSF.

[5] We also fit a series of models controlling for co-parent effects (e.g. effects of maternal AAB and interaction term while controlling for paternal AAB and interaction term). Parameter estimates were similar in magnitude to the single-parent models. Full results are available on https://osf.io/6c8jx/.

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References

Amad A, Ramoz N, Thomas P, Jardri R and Gorwood P (2014) Genetics of borderline personality disorder: systematic review and proposal of an integrative model. *Neuroscience & Biobehavioral Reviews* **40**, 6–19.

American Psychiatric Association (2013) *Diagnostic and Statistical Manual of Mental Disorders: DSM-5*. Washington, DC: American Psychiatric Association.

Bagge CL, Stepp SD and Trull TJ (2005) Borderline personality disorder features and utilization of treatment over two years. *Journal of Personality Disorders* **19**, 420–439.

Bandelow B, Krause J, Wedekind D, Broocks A, Hajak G and Rüther E (2005) Early traumatic life events parental attitudes family history and birth risk factors in patients with borderline personality disorder and healthy controls. *Psychiatry Research* **134**, 169–179.

Bates D, Mächler M, Bolker B and Walker S (2015) Fitting linear mixed-effects models using *lme*4. *Journal of Statistical Software* **67**, 1.

Belsky DW, Caspi A, Arseneault L, Bleidorn W, Fonagy P, Goodman M, Houts R and Moffitt TE (2012) Etiological features of borderline personality related characteristics in a birth cohort of 12-year-old children. *Development and Psychopathology* **24**, 251–265.

Bender DS, Dolan RT, Skodol AE, Sanislow CA, Dyck IR, McGlashan TH, Shea MT, Zanarini MC, Oldham JM and Gunderson JG (2001) Treatment utilization by patients with personality disorders. *American Journal of Psychiatry* **158**, 295–302.

Biskin RS (2015) The lifetime course of borderline personality disorder. *The Canadian Journal of Psychiatry* **60**, 303–308.

Bornovalova MA, Hicks BM, Patrick CJ, Iacono WG and McGue M (2011) Development and validation of the Minnesota Borderline Personality Disorder scale. *Assessment* **18**, 234–252.

Bornovalova MA, Hicks BM, Iacono WG and McGue M (2013) Longitudinal twin study of borderline personality disorder traits and substance use in adolescence: developmental change, reciprocal effects, and genetic and environmental influences. *Personality Disorders: Theory, Research, and Treatment* **4**, 23–32.

Bornovalova MA, Verhulst B, Webber T, McGue M, Iacono WG and Hicks BM (2018) Genetic and environmental influences on the codevelopment among borderline personality disorder traits, major depression symptoms, and substance use disorder symptoms from adolescence to young adulthood. *Development and Psychopathology* **30**, 49–65.

Brune M (2016) Borderline personality disorder: why 'fast and furious'? *Evolution, Medicine, and Public Health* **1**, 52–66.

Carpenter RW, Tomko RL, Trull TJ and Boomsma DI (2013) Gene-environment studies and borderline personality disorder: a review. *Current Psychiatry Reports* **15**, 336.

Carr S and Francis A (2009) Childhood familial environment, maltreatment and borderline personality disorder symptoms in a non-clinical sample: a cognitive behavioural perspective. *Clinical Psychologist* **13**, 28–37.

Chanen AM, Jackson HJ, McGorry PD, Allot KA, Clarkson V and Yuen HP (2004) Two-year stability of personality disorder in older adolescent outpatients. *Journal of Personality Disorders* **18**, 526–541.

Cohen P, Cohen J, Aiken LS and West SG (1999) The problem of units and the circumstance for POMP. *Multivariate Behavioral Research* **34**, 315–346.

Crowell SE, Baucom BR, McCauley E, Potapova NV, Fitelson M, Barth H, Smith CJ and Beauchaine TP (2013) Mechanisms of contextual risk for adolescent self-injury: invalidation and conflict escalation in mother–child interactions. *Journal of Clinical Child & Adolescent Psychology* **42**, 467–480.

Distel M, Trull T, Derom C, Thiery E, Grimmer M, Martin N, Willemsen G and Boomsma D (2008) Heritability of borderline personality disorder features is similar across three countries. *Psychological Medicine* **38**, 1219–1229.

Elkins IJ, McGue M and Iacono WG (1997) Genetic and environmental influences on parent–son relationships: evidence for increasing genetic influence during adolescence. *Developmental Psychology* **33**, 351–363.

Fonagy P and Luyten P (2009) A developmental, mentalization-based approach to the understanding and treatment of borderline personality disorder. *Development and Psychopathology* **21**, 1355–1381.

Frank H and Paris J (1981) Recollections of family experience in borderline patients. *Archives of General Psychiatry* **38**, 1031–1034.

Gignac GE and Szodorai ET (2016) Effect size guidelines for individual differences researchers. *Personality and Individual Differences* **102**, 74–78.

Golomb A, Ludolph P, Westen D, Block MJ, Maurer P and Wiss FC (1994) Maternal empathy, family chaos, and the etiology of borderline personality disorder. *Journal of the American Psychoanalytic Association* **42**, 525–548.

Grant BF, Chou SP, Goldstein RB, Huang B, Stinson FS, Saha TD, Smith SM, Dawson DA, Pulay AJ and Pickering RP (2008) Prevalence, correlates, disability, and comorbidity of DSM-IV borderline personality disorder: results from the Wave 2 National Epidemiologic Survey on Alcohol and Related Conditions. *The Journal of Clinical Psychiatry* **69**, 533.

Gratz KL, Latzman RD, Tull MT, Reynolds EK and Lejuez CW (2011) Exploring the association between emotional abuse and childhood borderline personality features: the moderating role of personality traits. *Behavior Therapy* **42**, 493–508.

Green P and MacLeod CJ (2016) *simr*: an R package for power analysis of generalized linear mixed models by simulation. *Methods in Ecology and Evolution* **7**, 493–498.

Hicks BM, Foster KT, Iacono WG and McGue M (2013) Genetic and environmental influences on the familial transmission of externalizing disorders in adoptive and twin offspring. *JAMA Psychiatry* **70**, 1076–1083.

https://doi.org/10.1017/S0033291719001260 Published online by Cambridge University Press

Hopwood CJ, Morey LC, Edelen MO, Shea MT, Grilo CM, Sanislow CA, McGlasha TH, Daversa MT, Gunderson JG, Zanarini MC, Markowitz JC and Skodol AE (2008) A comparison of interview and self-report methods for the assessment of borderline personality disorder criteria. *Psychological Assessment* **20**, 81–85.

Hunt E, Bornovalova MA and Patrick C (2015) Genetic and environmental overlap between borderline personality disorder traits and psychopathy: evidence for promotive effects of factor 2 and protective effects of factor 1. *Psychological Medicine* **45**, 1471–1481.

Iacono WG, Carlson SR, Taylor J, Elkins IJ and McGue M (1999) Behavioral disinhibition and the development of substance-use disorders: findings from the Minnesota Twin Family Study. *Development and Psychopathology* **11**, 869–900.

Jocklin V, McGue M and Lykken DT (1996) Personality and divorce: a genetic analysis. *Journal of Personality and Social Psychology* **71**, 288–299.

Johnson JG, Smailes EM, Cohen P, Brown J and Bernstein DP (2000) Associations between four types of childhood neglect and personality disorder symptoms during adolescence and early adulthood: findings of a community-based longitudinal study. *Journal of Personality Disorders* **14**, 171–187.

Johnson W, Krueger RF, Bouchard TJ and McGue M (2002) The personalities of twins: just ordinary folks. *Twin Research and Human Genetics* **5**, 125–131.

Kendler KS and Baker JH (2007) Genetic influences on measures of the environment: a systematic review. *Psychological Medicine* **37**, 615–626.

Kendler KS, Aggen SH, Czajkowski N, Røysamb E, Tambs K, Torgersen S, Neale MC and Reichborn-Kjennerud T (2008) The structure of genetic and environmental risk factors for DSM-IV personality disorders: a multivariate twin study. *Archives of General Psychiatry* **65**, 1438–1446.

Kendler KS, Myers J and Reichborn-Kjennerud T (2011) Borderline personality disorder traits and their relationship with dimensions of normative personality: a web-based cohort and twin study. *Acta Psychiatrica Scandinavica* **123**, 349–359.

Kernberg OF (2004) Borderline personality disorder and borderline personality organization: psychopathology and psychotherapy. In Magnavita JJ (ed.), *Handbook of Personality Disorders: Theory and Practice*. Hoboken, NJ: Wiley, pp. 92–119.

Keyes MA, Malone SM, Elkins IJ, Legrand LN, McGue M and Iacono WG (2009) The Enrichment Study of the Minnesota Twin Family Study: increasing the yield of twin families at high risk for externalizing psychopathology. *Twin Research and Human Genetics* **12**, 489–501.

Korzekwa MI, Dell PF, Links PS, Thabane L and Webb SP (2008) Estimating the prevalence of borderline personality disorder in psychiatric outpatients using a two-phase procedure. *Comprehensive Psychiatry* **49**, 380–386.

Linehan MM (1987) Dialectical behavior therapy for borderline personality disorder: theory and method. *Bulletin of the Menninger Clinic* **51**, 261–276.

Linehan MM (1993) *Cognitive-behavioral Treatment of Borderline Personality Disorder*. New York: Guilford.

Matteson LK, McGue M and Iacono WG (2013) Shared environmental influences on personality: a combined twin and adoption approach. *Behavior Genetics* **43**, 491–504.

McGue M, Keyes M, Sharma A, Elkins I, Legrand L, Johnson W and Iacono WG (2007) The environments of adopted and non-adopted youth: evidence on range restriction from the Sibling Interaction and Behavior Study (SIBS). *Behavior Genetics* **37**, 449–462.

Nash JC and Varadhan R (2011) Unifying optimization algorithms to aid software system users: *optimx* for *R*. *Journal of Statistical Software* **43**, 9.

Newman LK, Stevenson CS, Bergman LR and Boyce P (2007) Borderline personality disorder, mother–infant interaction and parenting perceptions: preliminary findings. *Australian & New Zealand Journal of Psychiatry* **41**, 598–605.

Paris J (2000) Childhood precursors of borderline personality disorder. *Psychiatric Clinics* **23**, 77–88.

Plomin R, DeFries JC and Loehlin JC (1977) Genotype-environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin* **84**, 309–322.

Purcell S (2002) Variance components models for gene–environment interaction in twin analysis. *Twin Research and Human Genetics* **5**, 554–571.

R Core Team (2018) R: *A Language and Environment for Statistical Computing*. Vienna, Austria: R Foundation for Statistical Computing. Available at http://www.r-project.org/.

Reichborn-Kjennerud T, Ystrom E, Neale MC, Aggen SH, Mazzeo SE, Knudsen GP, Tambs K, Czajkowski NO and Kendler KS (2013) Structure of genetic and environmental risk factors for symptoms of DSM-IV borderline personality disorder. *JAMA Psychiatry* **70**, 1206–1214.

Reichborn-Kjennerud T, Czajkowski N, Ystrom E, Orstavik R, Aggen SH, Tambs K, Torgersen S, Neale MC, Roysamb E, Krueger RF, Knudsen GP and Kendler KS (2015) A longitudinal twin study of borderline and antisocial personality disorder traits in early to middle adulthood. *Psychological Medicine* **45**, 3121–3131.

Reinelt E, Stopsack M, Aldinger M, Ulrich I, Grabe HJ and Barnow S (2014) Longitudinal transmission pathways of borderline personality disorder symptoms: from mother to child? *Psychopathology* **47**, 10–16.

Robins LM, Baber T and Cottler LB (1987) *Composite International Diagnostic Interview: Expanded Substance Abuse Module*. St. Louis: Authors.

Rohrer JM (2018) Thinking clearly about correlations and causation: graphical causal models for observational data. *Advances in Methods and Practices in Psychological Science* **1**, 27–42.

Rojas EC, Cummings JR, Bornovalova MA, Hopwood CJ, Racine SE, Keel PK, Sisk CL, Neale MC, Boker S and Burt SA (2014) A further validation of the Minnesota Borderline Personality Disorder Scale. *Personality Disorders: Theory, Research, and Treatment* **5**, 146–153.

Rojas EC, Hicks BM, Stark S, Hopwood CJ and Bornovalova MA (2015) Elaborating on the construct validity of the Minnesota Borderline Personality Disorder Scale (MBPD): a multi-sample, longitudinal examination. *Psychological Assessment* **27**, 332–339

Rosenstrom T, Torvik FA, Ystrom E, Czajkowski NO, Gillespie NA, Aggen SH, Krueger RF, Kendler KS and Reichborn-Kjennerud T (2018) Prediction of alcohol use disorder using personality disorder traits: a twin study. *Addiction* **113**, 15–24.

Scarr S and McCartney K (1983) How people make their own environments: a theory of genotype→environment effects. *Child Development* **54**, 424–435.

Shea TM, Edelen MO, Pinto A, Yen S, Gunderson JG, Skodol AE, Markowitz J, Sanislow CA, Grilo CM and Ansell E (2009) Improvement in borderline personality disorder in relationship to age. *Acta Psychiatrica Scandinavica* **119**, 143–148.

Silberschmidt A, Lee S, Zanarini M and Schulz SC (2015) Gender differences in borderline personality disorder: results from a multinational, clinical trial sample. *Journal of Personality Disorders* **29**, 828–838.

Soloff PH and Millward JW (1983) Psychiatric disorders in the families of borderline patients. *Archives of General Psychiatry* **40**, 37–44.

Spanier GB (1976) Measuring dyadic adjustment: new scales for assessing the quality of marriage and similar dyads. *Journal of Marriage and the Family* **51–52**, 15–28.

Spitzer RL, Williams JBW and Gibbon M (1987) *Structured Clinical Interview for DSM-III-R*. New York: Biometrics Research Department, New York State Psychiatric Institute.

Spotts EL, Neiderhiser JM, Towers H, Hansson K, Lichtenstein P, Cederblad M, Pederson NL and Reiss D (2004) Genetic and environmental influences on marital relationships. *Journal of Family Psychology* **18**, 107–119.

Stepp SD, Whalen DJ, Pilkonis PA, Hipwell AE and Levine MD (2012) Children of mothers with borderline personality disorder: identifying parenting behaviors as potential targets for intervention. *Personality Disorders: Theory, Research, and Treatment* **3**, 76–91.

Stepp SD, Whalen DJ, Scott LN, Zalewski M, Loeber R and Hipwell AE (2014) Reciprocal effects of parenting and borderline personality disorder symptoms in adolescent girls. *Development and Psychopathology* **26**, 361–378.

Tellegen A (1982) *Brief Manual for the Multidimensional Personality Questionnaire*. Unpublished manuscript, Minneapolis: University of Minnesota.

Tellegen A (2003) *Multidimensional Personality Questionnaire*. Minneapolis: University of Minnesota Press.

Tomko RL, Trull TJ, Wood PK and Sher KJ (2014) Characteristics of borderline personality disorder in a community sample: comorbidity, treatment utilization, and general functioning. *Journal of Personality Disorders* **28**, 734–750.

Trull TJ, Sher KJ, Minks-Brown C, Durbin J and Burr R (2000) Borderline personality disorder and substance use disorders: a review and integration. *Clinical Psychology Review* **20**, 235–253.

Weaver TL and Clum GA (1993) Early family environments and traumatic experiences associated with borderline personality disorder. *Journal of Consulting and Clinical Psychology* **61**, 1068.

White CN, Gunderson JG, Zanarini MC and Hudson JI (2003) Family studies of borderline personality disorder: a review. *Harvard Review of Psychiatry* **11**, 8–19.

Winograd G, Cohen P and Chen H (2008) Adolescent borderline symptoms in the community: prognosis for functioning over 20 years. *Journal of Child Psychology and Psychiatry* **49**, 933–941.

Winsper C, Zanarini M and Wolke D (2012) Prospective study of family adversity and maladaptive parenting in childhood and borderline personality disorder symptoms in a non-clinical population at 11 years. *Psychological Medicine* **42**, 2405–2420.

Zalewski M, Stepp SD, Scott LN, Whalen DJ, Beeney JF and Hipwell AE (2014) Maternal borderline personality disorder symptoms and parenting of adolescent daughters. *Journal of Personality Disorders* **28**, 541–554.

Zanarini MC (2000) Childhood experiences associated with the development of borderline personality disorder. *Psychiatric Clinics* **23**, 89–101.

Zanarini MC, Frankenburg FR, Dubo ED, Sickel AE, Trikha A, Levin A and Reynolds V (1998) Axis I comorbidity of borderline personality disorder. *American Journal of Psychiatry* **155**, 1733–1739.

Zanarini MC, Frankenburg FR, Hennen J and Silk KR (2004) Mental health service utilization by borderline personality disorder patients and Axis II comparison subjects followed prospectively for 6 years. *Journal of Clinical Psychiatry* **65**, 28–36.

Zanarini MC, Barison LK, Frankenburg FR, Reich DB and Hudson JI (2009) Family history study of the familial coaggregation of borderline personality disorder with Axis I and nonborderline dramatic cluster Axis II disorders. *Journal of Personality Disorders* **23**, 357–369.

Zweig F and Paris J (1991) Parent's emotional neglect and overprotection according to the recollections of patients with borderline personality disorder. *American Journal of Psychiatry* **148**, 648–651.

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