

MATERNAL TRANSMISSION OF BORDERLINE PERSONALITY DISORDER SYMPTOMS IN THE COMMUNITY-BASED GREIFSWALD FAMILY STUDY

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The authors longitudinally investigated the familial transmission of mothers' BPD symptoms to their offspring, taking maternal depression into consideration. The sample consisted of 323 offspring and their mothers from the community-based Greifswald Family Study. These families were examined for the first time when the children were about 15 years old (T_0), and again 5 years later (T_1), using self-ratings and interviews. Regression analyses revealed that maternal BPD symptoms and depression at T_0 were significant predictors of a number of BPD criteria that offspring met at T_1 . Furthermore, the analyses also predicted offspring's general psychopathology. In sum, the authors' findings provide evidence for familial aggregation of BPD symptoms and heightened levels of general psychopathology in offspring of mothers with high levels of BPD features, pointing to the need for providing early intervention for this high-risk group.

Borderline personality disorder (BPD) is characterized by a pervasive pattern of difficulties in emotion regulation, dissociative symptoms, self-destructive behavior, and unstable interpersonal relationships, leading to serious impairment in functioning (American Psychiatric Association, 2000;

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Due to our longitudinal design, this article is partly based on data also used in a previously published report (Barnow, Spitzer, Grabe, Kessler, & Freyberger, 2006).

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Barnow, Volker et al., 2009; Barnow et al., 2011; Lang et al., 2012). The prevalence rate in the general population ranges from 0.7% (Barnow, Stopsack, & Ulrich, et al., 2009; Coid, Yang, Tyrer, Roberts, & Ullrich, 2006) to over 5% (Grant et al., 2008). People suffering from BPD symptoms on a sub-threshold level are even more frequent (Sansone, Schumacher, Wiederman, & Routsong-Weichers, 2008; Trull, 1995) and also found to be impaired in various ways (Skodol et al., 2005; Trull, Useda, Conforti, & Doan, 1997).

Furthermore, borderline diagnoses as well as BPD symptoms were found to accumulate in families (Siever, Torgersen, Gunderson, Livesley, & Kendler, 2002; Silverman et al., 1991). Beside genetic influences (Distel et al., 2008), impaired interpersonal and family functioning could lead to said accumulation (Barnow, Stopsack, Grabe, et al., 2009; Newman & Stevenson, 2005). For example, mothers with BPD are insensitive toward their offspring (Crandell, Patrick, & Hobson, 2003) and often show disrupted affective communication with them (Hobson et al., 2009). In line with this finding, offspring of mothers with BPD (features) were found to be at risk for various psychopathological complaints (Weiss et al., 1996). For instance, in a former study we found a higher prevalence of emotional and behavioral problems in offspring of mothers with BPD than in children with other maternal diagnoses or healthy mothers (Barnow et al., 2006).

However, the transmissive pathways from parental BPD (symptoms) to offspring's complaints have not yet been fully clarified. Due to the high comorbidity between BPD and major depressive disorder (MDD) (Barnow, Herpetz, et al., 2007; Grant et al., 2008), and considering the fact that both of these disorders in parents lead to increased psychopathology in offspring (Lieb, Isensee, Hofler, Pfister, & Wittchen, 2002; Ulrich et al., 2011), common etiological factors have been proposed (Riso, Klein, Anderson, & Ouimette, 2000). This goes with the debate about whether BPD may represent an atypical form of Axis I disorder (Paris, Silk, Gunderson, Links, & Zanarini, 2009; Tyrer, 2009). Nevertheless, only a few studies examining the transmission of BPD (symptoms) have controlled for depressive disorders in parents (Barnow et al., 2006; Gasperini et al., 1991; Riso et al., 2000).

Another methodological problem is that most studies do not use community-based samples. Instead, inpatient or outpatient groups are investigated quite often (Baron, Gruen, Asnis, & Lord, 1985; Distel et al., 2008; Links, Steiner, & Huxley, 1988), thereby limiting the generalizability of the results. Furthermore, most studies are based on individuals meeting formal diagnostic criteria for BPD rather than on dimensional data (Feldman et al., 1995; Johnson et al., 1995; Weiss et al., 1996) and often cross-sectional designs are used (Gasperini et al., 1991; Herr, Hammen, & Brennan, 2008; Riso et al., 2000) instead of longitudinal approaches, which allow for a better examination of familial transmission. In addition, several studies obtained diagnostic information of BPD probands and their relatives exclusively (Baron et al., 1985; Zanarini, Gunderson, Marino, & Schwartz, 1988) or partially by using indirect interviews (Links et al., 1988; Riso et al., 2000), which could be subject to several biases.

In order to extend our above-mentioned findings, this study examined the familial transmission of BPD symptoms in a community-based sample using directly obtained dimensional data in a longitudinal design. Our first hypothesis was that the number of maternal self-rated borderline features at first assessment (T_0) would predict BPD symptoms in offspring about 5 years later (T_1). Second, we wanted to test whether mothers' depressive disorder at T_0 would also be a significant predictor for offspring BPD symptomatology. Furthermore, it should be tested whether maternal BPD symptoms would specifically lead to BPD symptoms in offspring or whether they increased psychopathological complaints in general.

METHOD

PARTICIPANTS

The sample of the current investigation was drawn from the population-based Greifswald Family Study (GFS; Barnow, Stopsack, Ulrich, et al., 2009; Barnow, Ulrich, Grabe, Freyberger, & Spitzer, 2007), a subpopulation of the Study of Health in Pomerania, Germany (SHIP; John et al., 2001). In SHIP, 4,308 people aged 20 to 79 were chosen at random between March 1997 and May 2000, proportional to the population size of each community, and stratified by age and gender. From this sample, 527 families who lived in a household with a minimum of one offspring between the ages of 11 and 18 years were asked to take part in the GFS. One hundred forty-one of these families could not be located or did not answer our phone calls and letters. In addition, 71 families refused to participate, resulting in a final sample of 315 families with whom assessments of parents ($n = 286$ mothers, mean age 39.8, $SD = 5.3$; $n = 194$ fathers, mean age 42.0, $SD = 5.4$) and offspring ($n = 381$, mean age 15.1, $SD = 2.3$) were conducted. There were no statistically significant differences between the 315 families included in the current study and the 212 nonparticipants in terms of the parents' education ($\chi^2 = 6.84$, $df = 7$, $p = .273$) and the number of children in the household ($t = -1.04$, $df = 526$, $p = .137$). Considering marital status, however, more parents of the investigated sample were married (75.6% vs. 63%, $\chi^2 = 9.99$, $df = 1$, $p = .002$).

Parents and offspring were again investigated about 5 years later between 2005 and 2008 (T_1). Most of the parents (85.6%) and offspring (87.7%; $n = 334$) took part in this follow-up. A total of 9 families ($n = 11$ offspring) were excluded from the current study due to missing data in one or more key variables relevant to this study, leaving 323 offspring (mean age 19.55, $SD = 2.43$, 44.9% males) with 247 biological mothers (mean age 44.47, $SD = 4.81$).

MATERIALS AND PROCEDURE

Assessments at T_0 . In mothers and offspring older than 16 years, BPD features were assessed using the self-rating part of the German version

of the Structured Clinical Interview for DSM-III-R (SCID-II; Wittchen, Schramm, Zaudig, & Unland, 1993). The borderline section consists of 13 items that can be answered "yes" or "no" and correspond to the eight BPD criteria in the DSM-III-R. As soon as at least one item for a corresponding criterion was answered with "yes," the criterion was taken as fulfilled. Number of fulfilled criteria (range 0 to 8) was the variable of interest in this study. Internal consistency for this subscale is $\alpha = 0.75$ and the retest reliability after 1 year is $r = 0.55$ and comparable to the reliability of personality dimensions in the five-factor model (Ball, Rounsaville, Tennen, & Kranzler, 2001). Due to diagnostic criteria (World Health Organisation, 1991), BPD features could not be assessed in children younger than 16 years.

Presence of depressive disorders in mothers was examined with the lifetime version of the standardized Munich-Composite International Diagnostic Interview (DIA-X/M-CIDI; Wittchen, Lachner, Wunderlich, & Pfister, 1998), an updated German version of the World Health Organization Composite International Diagnostic Interview (CIDI; Wittchen & Semmler, 1990) based on the research criteria of the ICD-10 and the *DSM-IV*. According to the authors, interrater reliability is high ($\kappa = .81-1.0$) and comparisons with clinical consensus diagnoses showed satisfying validities ($\kappa = .39-.82$), depending on interview section.

Assessments at T_1 . Personality disorder symptoms in mothers and offspring were examined using the SCID-II self-rating and interview for *DSM-IV* (Fydrich, Renneberg, Schmitz, & Wittchen, 1997). The nine self-rated BPD criteria were taken as fulfilled as soon as at least one corresponding item was answered with "yes." Psychopathological complaints in offspring were assessed with the German version of the Symptom Checklist-Revised (SCL-90-R; Derogatis, 1977; Franke, 1995), a self-rating inventory with nine clinical scales assessing exposure to particular symptoms during the past seven days. In their study, Hessel, Schumacher, Geyer, and Brähler (2001) found satisfying internal consistencies for the subscales, with Cronbach's alpha ranging from .75 to .88 in a large representative sample. According to Franke (1995), test-retest reliability is good, with values between $r = .69$ and $r = .92$, depending on subdimension and sample. Furthermore, the Global Severity Index (GSI) can be calculated as a measure of overall psychopathology (Derogatis, 1977).

Data Analysis. All analyses were performed using Predictive Analytics Software (PASW) version 19. First, bivariate correlations were calculated between maternal and offspring's BPD symptoms at both measurement points as well as maternal depression and offspring's general psychopathology, sex, and age.

In addition, we conducted a linear regression analysis predicting self-rated BPD symptoms in offspring at T_1 with maternal BPD features and depression at T_0 , controlled for offspring's sex, age, and initial BPD features in offspring that were at least 16 years old at T_0 . In addition, the influence of maternal BPD symptoms and depression on offspring's BPD interview ratings was examined using Poisson regression where BPD

symptoms of mothers and offspring are computed as count variables, because interview data violated conditions for linear regression. Due to the small number of offspring with mothers fulfilling more than six BPD criteria (6 criteria: $n = 6$, 7 criteria: $n = 12$, 8 criteria: $n = 0$), we aggregated them into one group. Last, we tested the effect of maternal BPD features and depression at T_0 on offspring's general psychopathology using linear regression analysis.

All analyses were reconducted using bootstrap techniques with 5,000 samples and for one child per mother only ($n = 247$) to account for non-independence of siblings.

RESULTS

First, as correlation analyses revealed (see Table 1), offspring's age and sex were significantly related to offspring's psychopathology, with younger and female persons reporting more symptoms. Therefore sex and age were included as control variables in further analyses. Also, maternal BPD symptoms and depression at T_0 were positively intercorrelated, and both were significantly associated to offspring's BPD features and general psychopathology at T_1 .

Accordingly, in the second step, maternal BPD symptoms and depression at T_0 were entered in linear regression analysis predicting self-rated BPD features in offspring about 5 years later, controlled for sex and age (see Table 2). Thereby, we found maternal BPD symptoms to be a significant predictor, whereas the diagnosis of a depressive disorder did not predict BPD symptomatology in offspring. Furthermore, as we additionally controlled for BPD features in offspring who were at least 16 years old at T_0 ($n = 165$), maternal self-rated BPD symptoms at T_0 still predicted offspring BPD symptomatology by trend ($\beta = 0.126$, $T = 1.697$, $p = .092$).

In Poisson regression analysis, maternal self-rated as well as offspring's fulfilled BPD criteria in the interview were taken as count variables. Therefore 85.1% of offspring did not fulfill any BPD criteria, whereas 2.8% fulfilled one, 3.7% two, 2.2% three, 1.9% four, 1.5% five, 1.2% six, 0.6% seven, 0.3% eight, and 0.6% fulfilled nine criteria. Model fit was good, with $\chi^2 = 84.43$, $p < .001$. Model effects were significant for maternal BPD features (Wald- $\chi^2 = 17.86$, $p = .007$), and depression at T_0 (Wald- $\chi^2 = 14.83$, $p < .001$), controlled for offspring's sex (Wald- $\chi^2 = 21.45$, $p < .001$) and age (Wald- $\chi^2 = 0.42$, $p = .516$). As visualized in Figure 1, maternal BPD was a significant predictor only when mothers fulfilled at least six BPD criteria at T_0 . Most of these mothers (83.3%) suffered from comorbid depression compared to a prevalence of 25.1% for depressive disorders in the whole sample (see also Table 1).

Furthermore, maternal depression at T_0 was a significant predictor of offspring severity of general psychopathology at T_1 , and maternal BPD symptoms predicted general symptom load by trend when controlled for offspring sex and age (see Table 2, and for better visualization see Figure 2).

TABLE 1. Descriptive Statistics and Correlations Between Maternal and Offspring's BPD Symptoms, as well as Maternal Depression and Offspring's Age, Sex, and Severity of General Psychopathology (GSI)

	Maternal T ₀			Offspring's					
	M	SD	Range	BPD Symptoms	Depression	T ₀ BPD Symptoms ^a	T ₁ BPD Symptoms SR	T ₁ BPD Symptoms INT	T ₁ GSI ^b
Offspring's age at T ₁	19.55	(2.43)	14-27	-0.05	ns	0.00	ns	-0.18 *	-0.13 *
Offspring's sex (% female) ^c	55.1%			0.09	ns	0.06	ns	0.13	0.15 **
Maternal T ₀									
BPD symptoms	2.35	(1.75)	0-7						
Depression ^d	25.1%	(n = 62)		0.35	***				
Offspring's									
T ₀ BPD symptoms ^e	3.45	(2.04)	0-8 ^a	0.22	**	0.14	t		
T ₁ BPD symptoms SR	2.71	(2.04)	0-9	0.18	**	0.13	*	0.40	***
T ₁ BPD symptoms INT	0.52	(1.50)	0-9	0.17	**	0.18	**	0.44	***
T ₁ GSI ^a	0.37	(0.35)	0-2,24	0.18	**	0.20	***	0.43	***

Notes. ^an = 165 (≥16 years old); ^bglobal severity index of general psychopathology as measured with the SCL-90-R; ^cmale = 0, female = 1; ^d0 = no, 1 = yes, 15.8% depressive episode, 6.9% recurrent depressive disorder, 4.5% dysthymia, 1.2% other depressive disorders; ns not significant, *p ≤ .10, **p ≤ .05, ***p ≤ .01, ****p ≤ .001.

TABLE 2. Linear Regression Analyses Predicting Number of Self-Rated Criteria That Offspring Fulfilled in the Borderline Section at T₁ as Well as Their General Psychopathology (GSI) with the Risk Factors Mothers' BPD Criteria Fulfilled in the Self-Rating and Mothers' Depression at T₀ Controlled for Offspring Sex and Age

	B	SE	Beta	T	p
Dependent variable: T ₁ offspring self-rated BPD symptoms					
Age	-0.21	0.04	-0.26	-4.84	***
Sex ^a	0.65	0.22	0.16	3.01	**
Mothers' depression at T ₀ ^b	0.39	0.27	0.08	1.45	ns
Number of criteria mothers fulfilled in borderline self-rating at T ₀	0.15	0.07	0.12	2.20	*
Dependent variable: T ₁ offspring general psychopathology (GSI) ^c					
Age	-0.02	0.01	-0.13	-2.47	*
Sex ^a	0.08	0.04	0.12	2.15	*
Mothers' depression at T ₀ ^b	0.13	0.05	0.16	2.74	**
Number of criteria mothers fulfilled in borderline self-rating at T ₀ ^b	0.02	0.01	0.11	1.83	^t

Notes. ^amale = 0, female = 1; ^b0 = no, 1 = yes; ^cglobal severity index of general psychopathology as measured with the SCL-90-R; ^t $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$.

When we reconducted all analyses using bootstrap techniques, the results remained stable. This was also the case when only one child per mother was included ($n = 247$) in order to avoid overweighting mothers with more than one offspring due to nonindependence of siblings.

DISCUSSION

In this study, we investigated the familial transmission of BPD symptoms from mother to child in a longitudinal design. Additionally, the role of maternal depressive disorders for the development of BPD features in offspring was examined. We also tested whether maternal BPD symptomatology would specifically lead to BPD symptoms in offspring or if psychopathology would increase in general.

First, we found that maternal self-rated BPD symptoms predicted BPD features in offspring about 5 years later. This finding is in line with other studies examining the transmission (Weiss et al., 1996) or familial aggregation of BPD features (Baron et al., 1985; Zanarini et al., 2004). In particular, our results show that BPD symptoms pass on from mother to child, even if the mother reported subthreshold BPD symptoms. When this transmission is examined in detail, offspring's fulfilled criteria in the interview were significantly predicted by maternal BPD symptomatology only when mothers met at least six criteria. Therefore, in accordance with biopsychosocial models, it can be concluded that the development of more severe BPD symptoms is based on a more serious familial predisposition than a less severe BPD psychopathology.

Furthermore, maternal depression also served as a predictor for offspring BPD psychopathology. Thus, both a high level of BPD symptoms in mothers and maternal depression led to BPD in offspring on a subthresh-

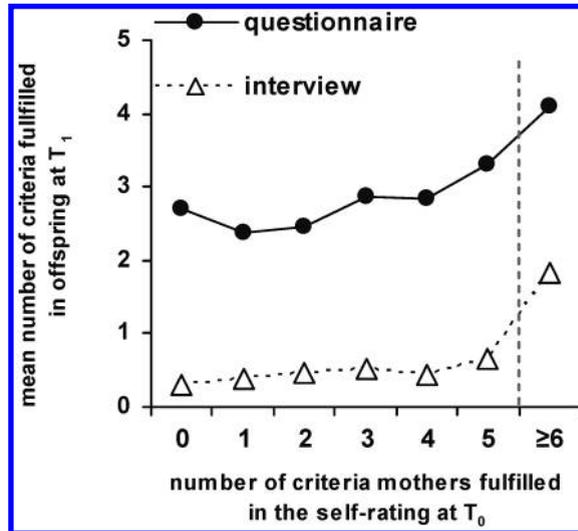


FIGURE 1. Mean numbers of criteria fulfilled in offspring in the borderline section of the SCID-II self-rating and interview at T₁ according to number of BPD criteria their mothers met in the questionnaire at T₀. Poisson regression coefficients for offspring’s BPD criteria fulfilled in the interview at T₁ as dependent variable: maternal symptoms = 0: b set to 0; 1 criterion: b = -0.073 (0.363), p = .841; 2 criteria: b = 0.110 (0.365), p = .763; 3 criteria: b = 0.110 (0.392), p = .780; 4 criteria: b = 0.049 (0.416), p = .907; 5 criteria: b = 0.481 (0.415), p = .246; ≥6 criteria: b = 0.923 (0.399), p = .021*.

old level. Regarding the high prevalence of comorbid depressive disorders in mothers meeting at least six BPD criteria in our sample, offspring with this familial background seem to constitute a high-risk group for the development of BPD symptoms. This result supports the suggestion of shared familial factors between BPD and MDD (Bandelow et al., 2005; Bellino et al., 2005; Zanarini, Barison, Frankenburg, Reich, & Hudson, 2009) with directly obtained data on Axis I and II disorders from both mothers and offspring—a set that can rarely be found in the literature on this topic (Links et al., 1988; Loranger, Oldham, & Tulis, 1982; Schulz et al., 1986).

Age also significantly influenced BPD symptoms in offspring, with younger children fulfilling more criteria than older ones. This age effect is in line with other studies, showing maturing of personality (Roberts, Caspi, & Moffitt, 2001) and a decrease of BPD traits from adolescence to adulthood (Bornovalova, Hicks, Iacono, & McGue, 2009).

In addition, besides BPD features in particular, severity of offspring’s general psychopathology was also predicted by maternal BPD symptoms and depression, which corresponds with studies showing higher psychopathology in children of mothers with BPD symptoms (Barnow, Spitzer, Grabe, Kessler, & Freyberger, 2006; Lewinsohn, Rohde, Seeley, Klein, & Gotlib, 2000). For example, Herr et al. (2008) reported a significant association between maternal BPD symptoms and interpersonal functioning,

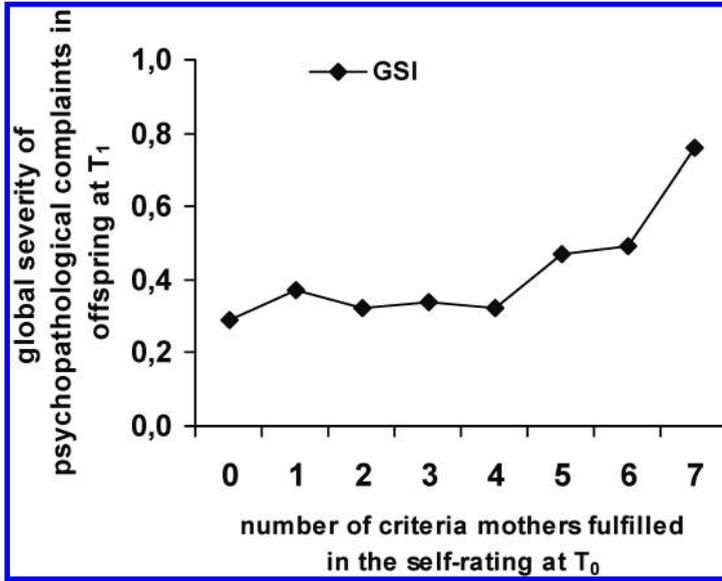


FIGURE 2. Offspring's psychopathological complaints (GSI) at T_1 according to number of BPD criteria their mothers met in the questionnaire at T_0 . None of the offspring had a mother who fulfilled eight criteria in the BPD self-rating.

attachment cognitions, and depressive symptoms in offspring. Thus, even though the number of BPD criteria met by offspring in our study is rather small, children of mothers with BPD symptoms and depression seem to be burdened with various other difficulties that can lead to impairment in everyday life. Winograd, Cohen, and Chen (2008) found in their community-based sample that subsyndromal BPD symptoms at the age of 14 had a negative impact on children's adult life in various areas, such as social functioning, life satisfaction, and academic attainment, in the subsequent 20 years.

These results add to our former study in which we also found numerous problems in children of mothers with BPD using a cross-sectional design (Barnow et al., 2006), but extend that study by showing that early complaints due to maternal BPD are substantially maintained during adolescence. Hence, our findings emphasize the importance of providing early programs to offspring of mothers with BPD symptoms, especially when maternal symptomatology is high and marked by comorbid depression, so as to diminish impairment in children and prevent progressive courses.

However, several limitations of our study need to be considered. First, we used self-rating data on maternal BPD features instead of interviews. This method is not unusual in studies assessing BPD symptoms (Herr et al., 2008; Sansone et al., 2008) and was not found to be less valid than interviews (Hopwood et al., 2008). Of course, these data reflect BPD on a subsyndromal level, but the literature has shown that persons who fulfill

only a few BPD criteria are also impaired (Skodol et al., 2005; Trull et al., 1997).

Second, we did not have sufficient data to include fathers in our analyses. One reason is the lack of availability of biological fathers in our sample due to divorce or separation. Furthermore, according to *DSM-IV* (American Psychiatric Association, 2000), 75% of the persons with BPD are female. It is therefore not surprising that there is considerable literature regarding mothers with BPD or the familial transmission of BPD via mothers (Crandell et al., 2003; Hobson et al., 2009), whereas fathers are often neglected. There is some evidence, however, linking paternal variables, for example, psychopathology, to offspring's BPD symptoms (Helgeland & Torgersen, 2004). In addition, the literature is inconsistent as to whether women are diagnosed as having BPD more frequently than men (Bjorklund, 2006) or whether prevalence may be equal among both sexes (Grant et al., 2008). Thus, the influence of BPD in fathers on offspring requires consideration in future research.

Third, we experienced a systematic loss of single mothers in our sample, so our results are not generalizable to this subgroup even though our ratios reflect the demographic distribution of single parents in the area of the former East Germany (Statistisches Bundesamt, 2010). In light of the fact that offspring of single mothers may have to face different and possibly greater challenges than children living with both parents (Bull & Mittelmark, 2009), future studies on the transmission of psychopathology should address these particular circumstances.

Nevertheless, there are numerous strengths to our study. First, we investigated a community sample in a longitudinal design in order to examine the familial transmission of BPD symptoms. According to the review by White, Gunderson, Zanarini, and Hudson (2003) on family studies in BPD and to the best of our knowledge, this is the first study that investigated familiarity of BPD dimensionally in a community sample, whereas in other studies categorical data of clinical samples were examined. Furthermore, we assessed BPD features and psychopathological complaints directly in mothers and in offspring, whereas other studies often had only indirect measurements for family members (White et al., 2003). In addition, we also examined mothers' depressive disorders in order to take the high comorbidity between MDD and BPD into account. Thus, our results can be generalized regarding mothers with multiple symptoms instead of BPD symptoms only, and therefore these results show higher ecological validity.

In sum, our findings provide evidence for familial aggregation of BPD symptoms and heightened levels of general psychopathology in offspring of mothers with high levels of BPD features, pointing to the need for providing early intervention for this high-risk group. Future research needs to consider the role of fathers in order to obtain a more comprehensive picture of the familial transmission and pathogenesis of BPD (symptoms) using longitudinal studies in community samples.

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