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Prospective associations between maternal borderline personality disorder symptoms, observed parenting and adolescent BPD features

Sophie Hodson
Manchester Metropolitan University
Jonathan Hill
Manchester Metropolitan University
Laura Bozicevic
University of Liverpool
Helen Sharp
University of Liverpool
Fay Huntley
University of Edinburgh
Evelyn Mary-Ann Antony
University of St Andrews
Margaret Owen
The University of Texas at Dallas
Nicky Wright
nicky.wright@mmu.ac.uk

Manchester Metropolitan University

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Abstract

Background

Limited evidence, entirely cross sectional and largely in infancy, links maternal borderline personality disorder (BPD) symptoms to lower quality of parenting. We conducted the first prospective study of maternal BPD symptoms and parenting, and tested the hypothesis that observed parenting mediates the association between maternal BPD symptoms and early adolescent BPD features.

Methods

Participants were members of a stratified subsample (N = 268) of the Wirral Child Health and Development Study (WCHADS), a prospective UK population-based cohort. Maternal DSM BPD symptoms were assessed by interview primarily during pregnancy, and maternal depressive symptoms by self-report up to age 3.5 years. Quality of parenting was rated from recorded mother-child conflict discussions at age 9.5 years, and child self-report of their own BPD features at age 12 years. Analyses were conducted using regression and structural equation models.

Results

Maternal BPD symptoms predicted lower supportive presence ($\beta = -0.22$, $p = 0.003$) and greater hostility ($\beta = 0.18$, $p = 0.008$), independently of maternal depressive symptoms. There was an association between maternal BPD symptoms and adolescent BPD features ($r = 0.18$, $p = 0.004$), which was markedly attenuated after adjustment for maternal depressive symptoms ($\beta = 0.09$, $p = 0.253$), and there was no evidence of mediation by quality of parenting.

Conclusions

Based on our findings, maternal BPD symptoms rather than depression, are associated with an enduring reduction in supportive presence and increase in hostility. While these dimensions of parenting are important for a range of mental health outcomes in children, they did not contribute to vulnerability to adolescent BPD features.

Introduction

Associations between maternal depression and quality of parenting have been widely replicated [1], but the role of personality dysfunction, which is strongly associated with depression, has received little attention. Similarly, little is known about the role of parental personality dysfunction in creating vulnerability to mental disorders in the next generation. This is surprising because over 40 years ago, researchers showed that the risk of emotional and behavioural problems in the children of psychiatric

patients was strongly associated with comorbid personality disorder [2]. If parental personality dysfunction contributes to both poorer parenting and offspring personality dysfunction, poorer parenting may represent one pathway through which risk for personality dysfunction is transmitted across generations. An examination of this hypothesis requires prospective measurement of parental personality dysfunction early in the child's life, parenting, and child BPD features. We describe the first study using such a prospective design to investigate the hypothesis.

Examining the role of personality dysfunction is impeded by limited consensus regarding the personality disorder construct [3]. This is evident in the discussions leading to, and arising from, the development of the DSM-V Alternative Model for Personality Disorders and the ICD-11-dimensional model [4–7]. Nevertheless, there is general agreement that there are no natural thresholds for personality disorder. This has major implications for the investigation of personality disorders, parenting and child mental health. While personality disorders above diagnostic thresholds are relatively rare in the general population (5–10%), personality disorder features are more common [8]. If personality disorder features below the diagnostic threshold are associated with lower quality parenting and with child mental disorders, they represent a much more common risk factor in the general population and hence in clinical samples. We focus in this paper on Borderline Personality Disorder (BPD) because its features including emotion dysregulation and interpersonal dysfunction are likely to affect parenting. BPD has also been the focus of several decades of psychological and biological research, including development of treatment modalities, with implications for treatment[9–12].

Two systematic reviews of the association between maternal BPD and parenting have been conducted, one focusing on diagnosis [13] and the other including dimensional assessment of BPD [14]. Both reviews concluded that maternal BPD is associated with lower sensitivity, greater hostility, intrusiveness and poorer emotional availability, but highlighted important methodological limitations, including small samples, clinical recruitment and a predominance of cross-sectional designs. In addition, many studies rely on questionnaire measures of parenting, raising the possibility of shared method variance when both maternal BPD and parenting are reported by the same individual. Self-report may be particularly problematic in BPD, where difficulties identifying and accurately reporting emotions and behaviour have been documented [15]. Observational studies of parenting have reported associations between maternal BPD diagnosis and symptoms and lower maternal sensitivity, emotional availability and autonomy support, alongside greater hostility, intrusiveness and disrupted parent-child interactions across infancy [16–21] and preschool and middle childhood [22–24], with limited evidence in adolescence from two small clinical comparison studies [25–26]. One of these found that mothers with BPD displayed lower promotion of autonomy and relatedness and greater hostility during observed interactions with their age 14–17-year-olds, after accounting for concurrent depression [25]. The other study reported greater severity of BPD symptoms was associated with fewer attachment-promoting behaviours and more invalidating and controlling parenting during observed conflict discussions with 10–15-year-olds[26]. Overall, the existing observational evidence has exclusively been cross-sectional and largely conducted in clinically ascertained samples. No previous study has examined whether maternal BPD symptoms assessed prenatally or early in a child's life predicts later parenting quality.

There is evidence that BPD symptoms are transmitted across generations. Twin and family studies indicate that BPD is moderately heritable [26–27]. Prospective studies of the relationship between maternal BPD symptoms when offspring were adolescents and subsequent BPD features in young people have reported modest associations [28–31]. Importantly all of these studies used a dimensional assessment of maternal BPD consistent with recent proposals for a dimensional approach [4–5]suggesting that variations below, as well as above, any diagnostic threshold may generate vulnerability.. As we noted earlier, the study of the role of parenting in the development of offspring's BPD features requires assessing parental BPD features early in life. No previous studies have examined linkages between parental BPD symptoms assessed prenatally, in infancy or in early childhood and later offspring BPD features.

Several studies have reported mediation analyses of parental BPD, quality of parenting and offspring psychopathology. However, most of them have been limited either by using offspring reports of parenting, which may be confounded by psychopathology [32–33], or by parental reports of their parenting and offspring outcomes and so susceptible to shared method variance [34]. To our knowledge, only one previous study has examined observed parenting as a mediator between maternal BPD symptoms and child mental health problems [24]. This was a cross-sectional study with measures of maternal BPD and child psychopathology assessed when children were between ages 5–12, and with number of psychiatric diagnoses as the outcome. The study found evidence of mediation by parenting, but the cross-sectional design precluded directional interpretations.

Early adolescence represents a particularly important developmental period in which to examine the intergenerational transmission of BPD. Borderline symptoms commonly emerge during adolescence [35–36], a period characterised by substantial developmental changes and an increasing vulnerability to mental health problems [37]. Historically it was argued that personality disorders could not be identified in adolescence as personality is not yet stable and assessment may be stigmatising [3]. However, research has indicated that the stability of BPD features from adolescence to adult life is no different to its stability across adulthood [38], that BPD in adolescence can be separated from other disorders in course and outcome [39] and is similar to adult BPD with respect to prevalence, risk factors and long-term outcomes [40–41]. In addition, the parent-child relationship undergoes significant reorganisation in adolescence as young people strive for greater autonomy while continuing to rely on caregivers for emotional support and regulation. This places new demands on parenting and may be particularly challenging for parents with BPD, whose interpersonal and emotion regulation difficulties could impair their ability to adapt to their child's increasing autonomy.

The current study examined whether maternal BPD symptoms assessed predominantly during pregnancy and early childhood predicted observed parenting in pre-adolescence (age 9.5 years) and whether supportive presence and parental hostility at that time mediated associations with later adolescent BPD features. We hypothesised that maternal BPD symptoms would predict lower supportive presence and greater hostility, that maternal BPD symptoms would be associated with adolescent BPD

features, and that supportive presence and hostility would mediate this association after adjustment for maternal depressive symptoms.

Methods

Participants

The sample consisted of members of a stratified subsample of the Wirral Child Health and Development Study, a prospective epidemiological sample of first-time mothers and their children born in 2007–2009. The overall cohort comprised 1233 women recruited in pregnancy from the sole antenatal care provider on the Wirral, with a live, singleton baby for long-term follow-up post-birth [42]. The Wirral includes areas spanning high to low socioeconomic deprivation but has limited ethnic diversity. The mean age at recruitment in pregnancy was 26.8 years (SD = 5.8, range 18–51), 41.8% of the sample were living in the most deprived quintile of UK neighbourhoods (indices of multiple deprivation, IMD) [34] and 96.1% were White British. The study used a two-stage stratified design in which a consecutive general population sample (the 'extensive' sample) is used to generate a smaller 'intensive' sample (N = 316) stratified by psychosocial risk (51% high risk) with more detailed measurement over time, and both are followed in tandem. The stratification was based on maternal responses to questions about psychological abuse in their current or recent partner relationship [43]. At age 5 a further stratum was added to the intensive sample [44] based on established thresholds for emotional or behavioural symptoms on the Child Behaviour Checklist (CBCL) [45] and the Antisocial Process Screening Device(APSD) [46–47]) data from age 3.5 years. This added 75 families to the intensive sample.

The study was approved by the Cheshire North and West Research Ethics Committee ethical approval for the data analysed here on 27/06/2006 (reference: 05/Q1506/107), 7/06/2010 (reference: 10/H1010/4), 22/12/2014 and 8/06/2020 (reference: 14/NW/1484). Mothers gave written informed consent and adolescents written informed assent.

The present analyses focus on 268 families who provided interview data in pregnancy (n = 215) or at age 5 (n = 53) assessing maternal BPD symptoms, self-report of chronic maternal depressive symptoms from 9-week post-birth to 3.5 years, observations of parent-child interaction at age 9.5 years and adolescent reported BPD features at age 12 years.

Comparisons between the analysis sample and the remainder of the eligible intensive sample (n = 142) indicated no significant differences in maternal BPD symptoms, chronic maternal depressive symptoms, child sex, deprivation, or sample stratification variables (p's all > 0.090). Mothers included in the analysis were slightly older at first pregnancy than those without complete data (mean age 27.55 SD = 6.15 years versus 26.31 SD = 5.77, p = 0.048).

Measures

Maternal BPD symptoms were assessed using a semi-structured diagnostic interview, the *Structured Clinical Interview for DSM-IV Axis II Disorders* (SCID-II) [48]. Mothers from the original intensive sample primarily completed this interview at 32 weeks gestation (n = 167) with some completing at the age 1 interview (n = 48), mothers who joined in the second stratification (n = 53) were interviewed at child age 5 years. Dimensional scores for maternal BPD symptoms were derived by summing the scores for each item: scored 0 (absent), 1 (subthreshold) and 2 (present) [49–50]. Ratings were made from audio recordings by two raters; interrater reliability based on 20 audio recordings was high (Intraclass correlation coefficient[ICC] = 0.91).

Adolescent BPD features were assessed via self-report at aged 12 using the 11-item *Borderline Personality Features Scale for Children* (BPFS-C-11) [51]. This 11-item version has been reported to have good criterion validity including against diagnostic interviews, internal reliability, and test-retest reliability [51–53]. The measure had good internal consistency in this sample (α = .86).

Parent-child interaction was assessed at age 9.5 years using the Family Issues Discussion task [53]. The parent and youth were instructed to choose three areas of disagreement to discuss (e.g., homework) from a set of 22 possible topics and asked to discuss each topic and try to resolve their differences. The 7-minute interactions were recorded and coded from videos using using a validated coding scheme developed for the National Institute of Child Health and Human Development Study of Early Child Care and Youth Development (NICHD-SECCYD) [54]. The scheme applies 7-point scales to rating items of parental behaviour, youth behaviour, and dyadic features of the interaction. Based on existing literature we focused on the maternal ratings of supportive presence, reflecting the extent to which mothers displayed positive regard, emotional support, involvement and affirmation of the child, and hostility, reflecting expressions of anger, criticism, rejection or discounting of the child or their ideas. Coders achieved good inter-rater reliability on 30 videos (ICC's = .70-.93).

Chronic maternal depressive symptoms were assessed via maternal report on the Edinburgh Postanatal Depression Scale (EPDS) [55] at age 9 weeks, 7 months, 14 months, 2.5 years and 3.5 years. The total scores were standardised and a mean taken across the five time points.

Other possible confounders included maternal age at first pregnancy, child sex recorded at birth (coded 0 = male, 1 = female) and family deprivation status. Deprivation was assessed using the 2019 IMD [56]. In this system, postcode areas in England are ranked from most to least deprived based on deprivation in seven domains: income, employment, health, education and training, barriers to housing and services, living environment and crime. Analysis used a binary variable reflecting 1 = most deprived quintile of UK neighbourhoods, 0 = all other quintiles.

Statistical analysis

All analyses were conducted in Stata version 19 [57]. Regression assumptions and model diagnostics were examined prior to analysis. Maternal BPD symptom scores were positively skewed, as expected in this population-based sample. Prior to analysis, distributions were inspected visually using histograms

and Q-Q plots, and influential observations were assessed using diagnostic statistics. No evidence of influential outliers was identified, and maternal BPD symptoms were therefore retained as a continuous variable in all analyses.

Bivariate associations between study variables were examined using Pearson's correlation coefficients. Preliminary linear regression analyses were conducted to examine the prospective associations between maternal BPD symptoms and each parenting and adolescent outcome variable prior to testing mediation models. Model 1 included maternal BPD symptoms only. Model 2 additionally adjusted for child sex, deprivation, maternal age at first pregnancy, the sampling stratification variables, and chronic maternal depressive symptoms.

The primary study hypothesis was examined using structural equation modelling with maximum likelihood estimation. A single mediation model simultaneously included observed supportive presence and hostility as mediators of the association between maternal BPD symptoms and adolescent BPD features. The model adjusted for child sex, neighbourhood deprivation, maternal age at first pregnancy, sampling stratification variables, and chronic maternal depressive symptoms. Direct, indirect and total effects were estimated, with indirect effects evaluated using the product-of-coefficients approach. Standardised regression coefficients (β), unstandardised beta with 95% confidence intervals and two-tailed *p* values are reported throughout.

Results

The descriptive statistics for the key study variables are shown in Table 1.

Table 1
Sample characteristics (N = 268)

Variable	**N**	**%**
Most deprived quintile	101	37.69
Adolescent Female sex	133	49.62
	Mean (SD)	Range
Maternal age at first pregnancy (years)	27.55 (6.15)	18–51
Maternal BPD symptoms	1.04 (2.24)	0–14
Chronic maternal depressive symptoms (z)	0.11 (0.85)	-1.14-3.97
Maternal supportive presence	4.52 (1.44)	1–7
Maternal hostility	2.18 (1.26)	1–6
Adolescent BPD features	24.57 (8.48)	11–53

Pearson correlations are presented in Table 2. Maternal BPD symptoms were moderately associated with chronic maternal depressive symptoms (r = 0.47, p < 0.001). Higher maternal BPD symptoms were associated with lower observed supportive presence (r=-0.23, p = 0.002), greater observed hostility (r = 0.16, p = 0.008), higher adolescent BPD features (r = 0.18, p = 0.004). Amongst the confounding variables; younger age at first pregnancy was associated with higher maternal BPD (r = 0.20, p=.001), higher chronic maternal depressive symptoms (r = 0.18, p=.008), lower supportive presence (r=-0.23, p<.001), higher hostility (r = 0.22, p < 0.001) and higher child BPD features (r = 0.13, p = 0.036). Greater deprivation was associated with higher maternal BPD (r = 0.16, p = 0.008) and higher chronic maternal depressive symptoms (r = 0.13, p = 0.037) and lower maternal age (r= -0.28, p < 0.001). Adolescent female sex was associated with lower hostility (r=-0.13, p = 0.034).

Table 2
Pearson correlations between key study variables

Variable	**1**	**2**	**3**	**4**	**5**	**7**	**8**
1. Maternal BPD symptoms							
2. Chronic maternal depressive symptoms	0.47***						
3. Supportive presence	-0.23**	-0.08					
4. Hostility	0.16**	0.04	-0.70***				
5. Adolescent BPD features	0.18**	0.16**	-0.10	0.09			
6. Younger maternal age	0.20**	0.18**	-0.23***	0.22***	0.13*		
7. Most deprived	0.16**	0.13*	-0.06	0.05	0.05	-0.28***	
8. Adolescent female	0.04	-0.01	0.01	-0.13*	0.05	-0.01	0.08
*p<.05 ** p<.01 *** p<.001							

Maternal BPD symptoms and observed parenting

Adjusted linear regression models demonstrated that higher maternal BPD symptoms were associated with lower supportive presence (β=-0.22, p = 0.003) and greater hostility (β = 0.18, p = 0.008). Chronic maternal depressive symptoms were not independently associated with either parenting dimension (p's > 0.22). Younger maternal age at first pregnancy was associated with lower supportive presence (β=-0.18, p = 0.003) and higher hostility (β = 0.21, p = 0.002). Child sex was associated with hostility, with mothers displaying lower hostility towards girls (β=-0.13, p = 0.033).

Table 3
Multiple linear regression models predicting observed parenting

Predictor	Supportive presence			Hostility		
	β	B (95% CI)	p	β	B (95% CI)	p
Maternal BPD symptoms	-0.22	-0.14 (-0.23, -0.05)	0.003	0.18	0.10 (0.03, 0.18)	0.008
Chronic maternal depressive symptoms	0.08	0.14 (-0.09, 0.37)	0.240	-0.80	-0.12 (-0.31, 0.07)	0.223
Younger maternal age	-0.18	-0.04 (-0.02, -0.07)	0.003	0.21	0.04 (0.02, 0.07)	0.002
Sex	-0.01	-0.01 (-0.34, 0.33)	0.937	-0.13	-0.31 (-0.60, -0.03)	0.033
Most deprived	0.02	0.07 (-0.31, 0.45)	0.711	-0.04	-0.04 (-0.37, 0.29)	0.825
Stratification factor 1	0.02	0.12 (-0.51, 0.75)	0.712	-0.01	-0.01 (-0.63, 0.61)	0.985
Stratification factor 2	-0.05	-0.18 (-0.60, 0.24)	0.406	-0.08	-0.25 (-0.60, 0.10)	0.166
Stratification factor 3	-0.09	-0.32 (-0.79, 0.16)	0.193	0.06	0.19 (-0.24, 0.61)	0.539

Maternal BPD symptoms and adolescent BPD features

After adjustment for covariates, maternal BPD symptoms were not independently associated with adolescent BPD features (β = 0.09, p = 0.253). Chronic maternal depressive symptoms showed a trend towards an association with adolescent BPD features (β = 0.10, p = 0.156), whereas maternal age at first pregnancy, child sex, socioeconomic deprivation and the sampling stratification variables were not significant predictors.

Table 4
Multiple linear regression models predicting adolescent borderline features

Predictor	β	B (95% CI)	p
Maternal BPD symptoms	0.09	0.35 (-0.25, 0.96)	0.253
Chronic maternal depressive symptoms	0.10	0.97 (-0.39, 2.33)	0.156
Younger maternal age	−0.09	-0.12 (-0.30, 0.07)	0.211
Child sex	0.04	0.69 (-1.38, 2.76)	0.507
Most deprived	−0.01	-0.10 (-2.24, 2.05)	0.931
Stratification factor 1	0.07	2.05 (-1.53, 5.63)	0.261
Stratification factor 2	0.05	0.96 (-1.81, 3.74)	0.494
Stratification factor 3	0.01	0.13 (-2.79, 3.04)	0.931

Mediation analyses

A structural equation model was used to examine whether observed supportive presence and hostility jointly mediated the bivariate association between maternal BPD symptoms and adolescent BPD features. Maternal BPD symptoms were independently associated with lower supportive presence (β=-0.20, p = 0.001) and greater hostility (β = 0.18, p = 0.034). However, neither supportive presence (p = 0.808) nor hostility (p = 0.634) was associated with adolescent BPD features. The direct association between maternal BPD symptoms and adolescent BPD features also remained non-significant (p = 0.383). Consistent with these findings, there was no evidence that either supportive presence (p = 0.839) or hostility (p = 0.570) mediated the association between maternal BPD symptoms and adolescent BPD features.

Table 4
Structural equation model examining the indirect effects of maternal BPD symptoms on adolescent BPD features through observed parenting

Path	β	B (95% CI)	p
Predicting supportive parenting			
Maternal BPD → Supportive presence	-0.22	-0.14 (-0.23, -0.06)	0.001
Predicting hostility			
Maternal BPD → Hostility	0.18	0.10 (0.03, 0.18)	0.034
Predicting adolescent BPD			
Maternal Supportive presence → Adolescent BPD	-0.02	-0.11 (-1.21, 0.98)	0.808
Maternal Hostility → Adolescent BPD	0.05	0.36 (-0.86, 1.58)	0.634
Maternal BPD → Adolescent BPD (direct)	0.08	0.30 (-0.29, 0.89)	0.383
Indirect effects			
Via supportive presence		0.02 (-0.14, 0.17)	0.839
Via hostility		0.04 (-0.09, 0.17)	0.570

Discussion

Maternal BPD symptoms assessed primarily during pregnancy and early childhood prospectively predicted lower supportive presence and greater hostility in an issues-discussion task in pre-adolescence, independent of chronic maternal depressive symptoms. Maternal BPD symptoms were associated with adolescent BPD features at the bivariate level; however, this association was attenuated and no longer statistically significant after adjustment for maternal depressive symptoms and other covariates. There was no evidence that either supportive presence or hostility mediated the association between maternal BPD symptoms and adolescent BPD features.

These findings build on previous systematic reviews of entirely cross-sectional studies of infants which concluded that BPD is associated with lower sensitivity and more hostile, intrusive and emotionally unavailable parenting than comparison mothers [13–14]. They extend the limited existing observational studies of adolescents or pre-adolescents which to date were conducted in clinical samples and report lower promotion of autonomy and relatedness and greater hostility during mother-adolescent interactions [25] and fewer attachment-promoting behaviours alongside more invalidating and controlling parenting [26]. Unlike previous studies, maternal BPD symptoms in the current study were assessed prospectively, predominantly during pregnancy and early childhood and before the assessment of parenting, reducing the potential for reciprocal influences of child characteristics on maternal BPD symptoms. Furthermore, the association between maternal BPD symptoms and observed parenting remained after accounting for maternal depressive symptoms, which were not associated with

parenting, suggesting that these parenting differences may reflect the interpersonal dysfunction and emotion regulation difficulties that are central features of BPD.

Although maternal BPD symptoms showed a small and significant association with adolescent BPD features at the bivariate level, this association was attenuated and no longer statistically significant after adjustment for maternal depressive symptoms and other confounders. This was the first study with assessments of BPD during pregnancy and early childhood with follow up to adolescent BPD features and so there is no direct comparison with existing evidence. However, studies with assessments of maternal BPD symptoms when children were adolescents and follow up to later adolescence or adulthood have reported somewhat larger associations [29–31].

We interpret this difference cautiously because the differences in effect size were not large. However, processes in the period intervening between early childhood and adolescence on the one hand, and between mid-adolescence and adulthood on the other are likely to be different. Furthermore, BPD symptoms as assessed in this study at 12 years may represent emerging borderline features that are less stable and potentially less differentiated than the more established borderline pathology assessed in later adolescence and adulthood [58]. Together, these findings suggest that the strength of intergenerational associations may vary across development and highlight the importance of examining the transmission of BPD symptoms across multiple developmental stages using repeated assessments of both maternal and offspring psychopathology.

Contrary to our hypothesis, there was no evidence that observed supportive presence or hostility mediated the association between maternal BPD symptoms and adolescent BPD features. As we noted earlier all previous studies of mediation have suffered from limitations arising from adolescent-reported parenting or cross-sectional design. By contrast, the present study assessed maternal BPD symptoms predominantly during pregnancy and early childhood, observed parenting approximately nine years later, and assessed offspring BPD symptoms three years after the parenting observation, allowing a clearer examination of the prospective association between maternal BPD, later parenting and offspring BPD symptoms. The present findings are also consistent with growing evidence that, although parental BPD prospectively predicts parenting, these broad dimensions of parenting may have limited prospective associations with offspring BPD during adolescence. One previous study found that adolescent girls' BPD symptoms predicted subsequent changes in parenting, whereas parenting did not predict subsequent changes in BPD symptoms [57].

In addition, this study examined broad dimensions of parenting quality. Developmental models of BPD emphasise more specific interpersonal processes, including emotional invalidation, difficulties supporting children's emotion regulation, impairments in parental mentalising, and challenges negotiating attachment and autonomy across development [59]. A distinguishing feature of parenting in mothers with BPD may be oscillation between hostile, controlling behaviour and emotional withdrawal or passive aloofness, rather than persistently low support or high hostility [58]. These dynamic and inconsistent interaction patterns are not well captured by global ratings. Future prospective studies

should examine more specific and reciprocal interpersonal processes to better understand how vulnerability to BPD is transmitted across generations.

The present study has several important strengths. Maternal BPD symptoms were assessed prospectively, predominantly during pregnancy and early childhood and prior to the assessment of parenting and offspring outcomes. The study also used observational assessment of parenting, which removed possible bias from shared method variance associated with self-report measures and enabled the assessment of parenting behaviours that may not be accurately perceived or reported by parents or adolescents. Finally, the repeated assessment of maternal depressive symptoms across childhood enabled adjustment for maternal depressive symptoms, allowing examination of whether associations between maternal BPD and parenting were independent of this highly comorbid condition.

The study also has several limitations. First, although the sample was relatively large for an observational study, it may have been underpowered to detect small effects. However, there was little evidence of an association between observed parenting and adolescent BPD features, suggesting that the absence of mediation was unlikely to reflect statistical power alone. Second, parenting was assessed during a single structured conflict discussion, which may not fully capture parenting behaviours across everyday contexts. Third, although the WCHADS cohort is broadly representative of the population from which it was recruited, the Wirral has relatively high levels of socioeconomic deprivation and limited ethnic diversity compared with the wider UK population.

Although observed supportive presence and hostility did not explain the association between maternal and offspring BPD symptoms, the finding that maternal BPD symptoms assessed predominantly during pregnancy and early childhood prospectively predicted parenting almost a decade later has important clinical implications. In the UK, substantial investment in perinatal mental health services has been strongly influenced by a concern about the effects of postnatal depression, on children's mental health and several trials have been motivated by the same concern [60]. These findings suggest that elevated BPD symptoms identify a group of mothers who may experience persistent difficulties in parent-child interactions extending well beyond infancy. Early identification of mothers with elevated BPD symptoms may therefore provide an opportunity to offer interventions that strengthen parenting and support parent-child relationships across childhood, rather than focusing exclusively on the perinatal period. The absence of mediation also has important implications for intervention development. The findings suggest that improving general parenting behaviours such as support and reducing hostility may not, in isolation, be sufficient to reduce intergenerational transmission of BPD symptoms. Instead, interventions may need to target parenting processes that may be more closely aligned with borderline pathology. Future longitudinal studies incorporating repeated assessments of both parenting and offspring psychopathology will be important for identifying the developmental mechanisms through which vulnerability to BPD is transmitted across generations and for informing more targeted preventive interventions.

Declarations

Data availability statement: Due to ethical constraints supporting data cannot be made openly available. Supporting data are available to bona fide researchers on approval of an application for access. Further information about the data and conditions for access are available at the University of Liverpool Research Data Catalogue: https://doi.org/10.17638/datacat.liverpool.ac.uk/564.

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Author Contribution

SH, NW, JH and HS conceptualised the study. HS and JH designed the original cohort study and acquired funding. NW and FH contributed to data collection. LB, SH, MO and FH conducted the observational coding of parent-child interactions. SH and NW drafted the manuscript. All authors reviewed and critically revised the manuscript and approved the final version.

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