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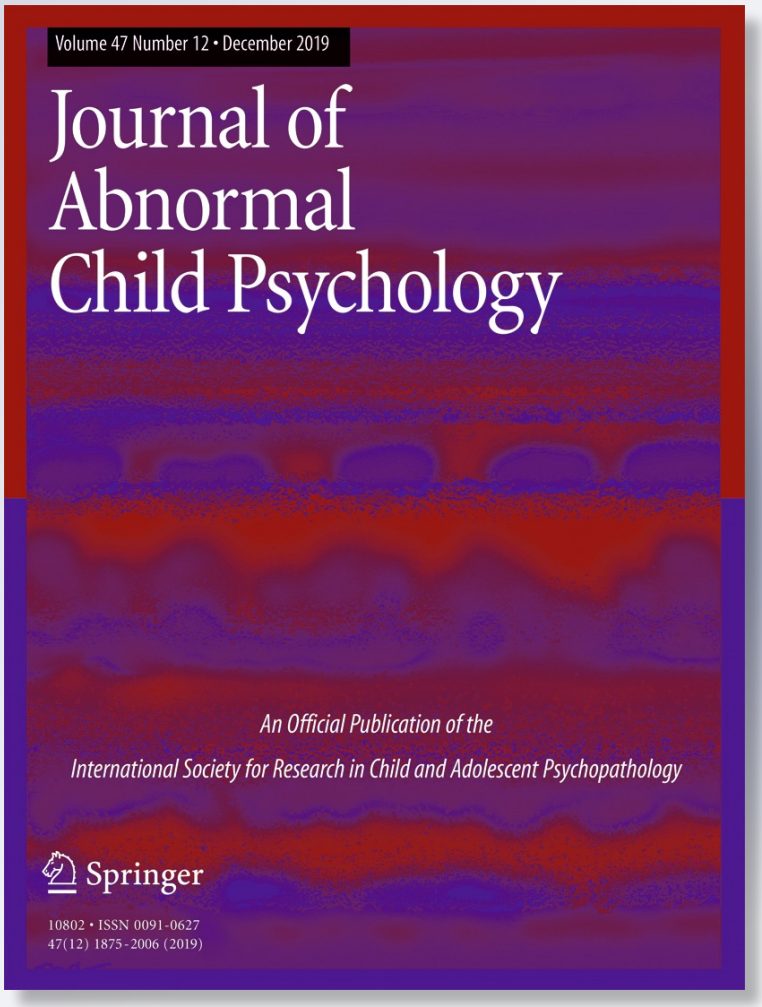

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Parents' Personality-Disorder Symptoms Predict Children's Symptoms of Anxiety and Depressive Disorders – a Prospective Cohort Study

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Abstract

Personality disorder (PD) symptomatology is characterized by interpersonal problems and emotional dysregulation, which may affect offspring of parents with PD symptoms. Notably though, studies are needed to discern (i) whether parental PDs *forecast* symptoms of psychiatric disorders in offspring during their childhood years and (ii) whether such prospective relations obtain after accounting for common causes (e.g., genetics, common methods). To address these issues, we followed up a community sample of Norwegian children biennially from ages 4 to 8 ($n = 594$), using a semi-structured psychiatric interview (PAPA/CAPA) to capture DSM-IV defined symptoms of emotional disorders. Parental symptoms of personality disorders were captured by the DSM-IV and ICD-10 Personality Questionnaire (DIP-Q), whereas depression and anxiety in caregivers were measured using the Beck Depression Inventory –II and Beck Anxiety Inventory, respectively. Upon applying a hybrid fixed and random effects method that takes into account all unmeasured time-invariant confounders, we found that: (i) Parental symptoms of DSM-IV defined Cluster A and C were related to symptoms of anxiety disorders in offspring two years later, even after accounting for children's initial levels of anxiety and parental anxiety, whereas (ii) Parental DSM-IV Cluster B predicted symptoms of depressive disorders in children, adjusted for children's initial levels of depression and parental depression. Clinical implications of the results are discussed.

Keywords Personality disorder · Anxiety · Depression · Symptoms · Parents · Children

Introduction

Children of parents suffering from depression and anxiety are at elevated risk of developing anxiety and depression (S. H. Goodman et al. 2011; Micco et al. 2009). Notably, similar evidence is rather limited in the case of parental personality disorders (PDs), despite these being pervasive conditions—characterized by persistent dysfunctional interpersonal patterns, unstable emotional regulation, poor impulse control, affective lability, and deviant cognitive-affective modulation—that are likely to undermine child well-being. Cross-sectional studies confirm that PD symptoms in parents are associated with poor mental health in children (Barnow et al. 2006; Berg-Nielsen et al. 2002; Berg-Nielsen and Wichstrom 2012; Bertino et al. 2012; Weiss et al. 1996). The present inquiry extends such prior research, evaluating whether parental PDs *forecast* symptoms of psychiatric disorders, specifically anxiety and depression, in offspring during their childhood years.

Why Parental PDs Should Affect Child Psychopathology

Embedded in a developmental psychopathological framework (Sroufe and Rutter 1984) attachment theory anticipates a link

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between problematic parenting/parent-child interaction (e.g., insecure attachment, restriction, overprotectiveness) and emotional problems in children (Zahn-Waxler et al. 2000), one for which there is empirical support (Cassidy and Berlin 1994; Chorpita and Barlow 1998; McLeod et al. 2007a; Ballash et al. 2006; Rapee 1997; Rapee et al. 2009). Consistent with Belsky's (1984; Belsky and Jaffee 2006) model of the determinants of parenting, evidence indicates that parents with PD symptoms engage in more problematic parenting (e.g., poor communication, limited praise/encouragement, inconsistent parental discipline) than those without such symptoms (Johnson et al. 2006; Wilson and Durbin 2012); it is via this adverse impact on parenting and family life that parental PDs are thought to affect children (Berg-Nielsen et al. 2002). In line with this view, Gratz and associates (Gratz et al. 2014) found that mothers with borderline PD had infants who displayed emotion regulation difficulties, and the relation between maternal PD and the infant's functioning was mediated by the mother's emotional dysfunction (Gratz et al. 2014).

To date, three prospective studies provide some evidence of the predictive power of parental PDs. Conroy et al. (2012) reported that maternal postpartum PD assessed when children were two months old predicted dysregulation but not internalizing or externalizing problems in 18-month old infants. Relatedly, Davies and associates (Davies et al. 2012) observed that maternal antisocial PD assessed when children were two years of age forecast disruptive behavior in offspring one year later. In a group of psychiatric patients and their children (aged under 15) followed yearly over a period of 4 years, Rutter and Quinton (1984) found that children of parents with PDs were more likely to develop mental health problems than children of parents with any other diagnoses investigated (i.e., affective disorders and psychosis). Methodological issues regarding these three studies should be highlighted. Because all focused on parents with a diagnosed PD, samples were relatively modest in size ($n \leq 200$). Notable as well is that child problems were measured using teacher- or parent-completed checklists, the accuracy of which is limited when it comes to assessing DSM-defined pathology (Sveen et al. 2013).

Parental PDs are likely to increase the risk of maladaptive development in offspring in several ways, including (i) impaired ability to support the development of well-functioning emotion regulation; (ii) impaired parenting abilities; and (iii) by exposing the child to heightened levels of interpersonal and family conflict, as such conflicts are more likely when one of the partners display PD symptoms (Whisman and Schonbrun 2009; South et al. 2008). In a dimensional approach to PDs, which is applied herein, PDs can be seen as maladaptive variants of the personality traits described in the Five-Factor Model of Personality (FFM) (Widiger et al. 2002), an assumption that has been empirically corroborated (Samuel et al. 2013; Trull and Durrett 2005). Consider in this regard a meta-analytic review focused on the FFM (DeYoung et al. 2007; McCrae and Costa Jr 2013) by Prinzie and colleagues (Prinzie et al. 2009) which indicated that warm, responsive parenting and autonomy support were negatively related to Neuroticism. People high in Neuroticism are more likely to be emotionally unstable, be easily distressed, tense, nervous and anxious, characteristics reminiscent of the "anxious-fearful" or Cluster C PDs. Further, although some common features of personality traits are seen across most of the PDs, FFM traits have also been found to distinguish between different disorders (Trull and Durrett 2005; Fowler et al. 2015).

Specific Relations between Parental PDs and Emotional Symptoms in Offspring

Given the link between personality traits and parenting and the heterogeneity in personality traits within specific PDs (Fowler et al. 2015; Henriques-Calado et al. 2014), it is noteworthy that Wilson and Durbin (2012) found that even though some dysfunctional parent-child interactions were similar across all PDs, others seemed to be specific to particular PDs. For example, the DSM-IV defined Cluster B, constituting antisocial, borderline, histrionic, and narcissistic PDs and known as the "dramatic-emotional-erratic" cluster, was associated with reduced parental responsiveness. In contrast, both Cluster A, characterized by paranoid, schizoid and schizotypal PDs and known as the "odd-eccentric" cluster, and Cluster C, characterized by avoidant, dependent, and obsessive-compulsive PDs and known as the "anxious-fearful" cluster, was associated with more frequent attempts to influence or control the child. Such findings clearly raise the prospect, as does work using the FFM, that different PD symptomatology may affect children differently, a proposition evaluated in the research reported herein.

Theoretical models stipulate that parental over-control may increase the risk for childhood anxiety by reducing the child's opportunity to develop his/her autonomy, as well as by communicating to the child that events are outside the child's control, thereby undermining a sense of self-efficacy which is associated with increased vulnerability for anxiety disorders (Chorpita and Barlow 1998). Further, cognitive models of anxiety suggest that socialization experiences (e.g. parental overprotection) may sensitize children to anxiety-provoking situations and contribute to the development of a biased attention to signals of threat or danger (Zahn-Waxler et al. 2000; Beck and Clark 1997). Consistent with this analysis is evidence that parental influence, control and overprotection, parenting characteristics associated with Cluster A and C PDs (Wilson and Durbin 2012), are specifically related to anxiety in children (Beesdo et al. 2010; Rapee 1997; McLeod et al. 2007b; Feng et al. 2008).

Childhood depression is also associated with parental control, but more strongly so to parental acceptance/rejection and limited warmth (Beesdo et al. 2010; McLeod et al. 2007a),

which accords well with attachment theory. These observations, in concert with the preceding discussion of personality and parenting, lead us to hypothesize that because parental acceptance and emotional warmth are related to parent responsiveness (Darling and Steinberg 1993), and parental Cluster B symptoms are associated with reduced responsiveness (Wilson and Durbin 2012), that Cluster B symptoms may specifically affect children's risk of depression. Consistent with this claim is evidence that paternal antisocial behavior (Cluster B) is associated with depression in adolescents (Marmorstein et al. 2004).

Is the Relation between Parental PD and Offspring Psychopathology Due to Confounding?

There is a real possibility that observed associations between parent and child phenotypes, even prospective ones and those seemingly mediated by parenting, could be fully or partly explained by genes shared by parents and children (Kendler et al. 2008; Livesley et al. 1998; Rutter et al. 2001). The impact of confounders other than genetics also needs to be entertained, including common-method effects (e.g., parents reporting on their own and their children's symptoms), overlapping symptoms across disorders, and stable environmental factors influencing both symptoms of parental PD and children's emotional disorders (e.g., socio-economic disadvantage, chronic marital conflict). Fortunately, fixed effects models hold the promise of illuminating longitudinal relationships while adjusting for all time-invariant unmeasured confounders (e.g., genetics) (Firebaugh et al. 2013; Allison 2009; Bollen and Brand 2010). In consequence, they are regarded as a novel and groundbreaking approach for investigating developmental psychopathology (Klein et al. 2017). Indeed, for these reasons, we adopted this method in the current observational study, an approach that has recently been successfully applied to investigate the development of psychopathology in children (Wichstrom et al. 2017b, a).

Summary and Objectives

Due to their enduring nature and the relationship-difficulties they entail, parental PDs are hypothesized to increase the risk of mental health problems in offspring. The current study is the first to *prospectively* examine whether Cluster A, B and C symptoms are predictive of children's DSM-IV defined symptoms of anxiety and depressive disorders, using a large sample of Norwegian 4-year olds followed up at ages 6 and 8 years using a fixed effects approach. Based on the above theorizing and empirical findings we hypothesize that DSM-defined symptoms of anxiety when children are 6 and 8 years of age will be predicted by parental Cluster A and C symptoms two years earlier (i.e., children aged 4 and 6, respectively), adjusted for baseline levels of children's anxiety, parental anxiety and all time-invariant unmeasured confounders. Children's symptoms of major depression, on the other hand, we expect to be predicted by parental Cluster B symptoms, after adjusting for parental depression, prior child depression, and unmeasured time-invariant factors.

Method

Procedure and Participants

All children born in 2003 and 2004 in the city of Trondheim, Norway and their parents were invited to participate in this study by a letter sent to their homes. The letter included a screening assessment for children's emotional and behavioral problems (The Strengths and Difficulties Questionnaire (SDQ) version 4–16) (R. Goodman et al. 2000), which parents completed and brought to the community health check-up when children were 4 years old. There, they received information about the study from the health nurse, who also obtained written participant consent. As shown in Fig. 1, almost everyone who was eligible to participate in the study appeared at the health clinic. Thus, the sample was essentially a community sample. Insufficient proficiency in Norwegian to fill out the SDQ scheme was used as criterion for exclusion.

To increase variability and thus statistical power, we oversampled for mental health problems in the children: Children were allocated to four strata according to their SDQ scores (cut-offs: 0–4, 5–8, 9–11, and 12–40), and the probability of selection increased with increasing SDQ scores (0.37, 0.48, 0.70, and 0.89 in the four strata, respectively). Out of 1250 invited families, 997 (79.8%) were interviewed and tested at time 1 (T1) (Fig. 1). The mean age of the children (49.1% male, 50.9% female) at T1 was 4.4 years (SD = 0.21); 84.4% of informants were mothers; the majority of participating parents were ethnic Norwegians (93% of the mothers and 91% of the fathers), and 89.1% were married or cohabitating. The sample was comparable with the Norwegian parent population with regard to parents' level of education (Statistics Norway 2012): 5.7% of the informants were managers, 25.7% were higher level professionals, 39% were lower-level professionals, 26% were formally skilled workers, 0.5% were farmers/fishermen, and 3.1% were unskilled workers.

Subsequent waves of assessment took place two (T2, n = 795; M_{age} = 6.7 years, SD = 0.17) and four years later (T3, n = 699; M_{age} = 8.8 years, SD = 0.24). In some cases, the parent accompanying the child was not the same at T1 and T2. We therefore only examined cases in which the same parent reported on PD at the two time-points (n = 594). The Regional

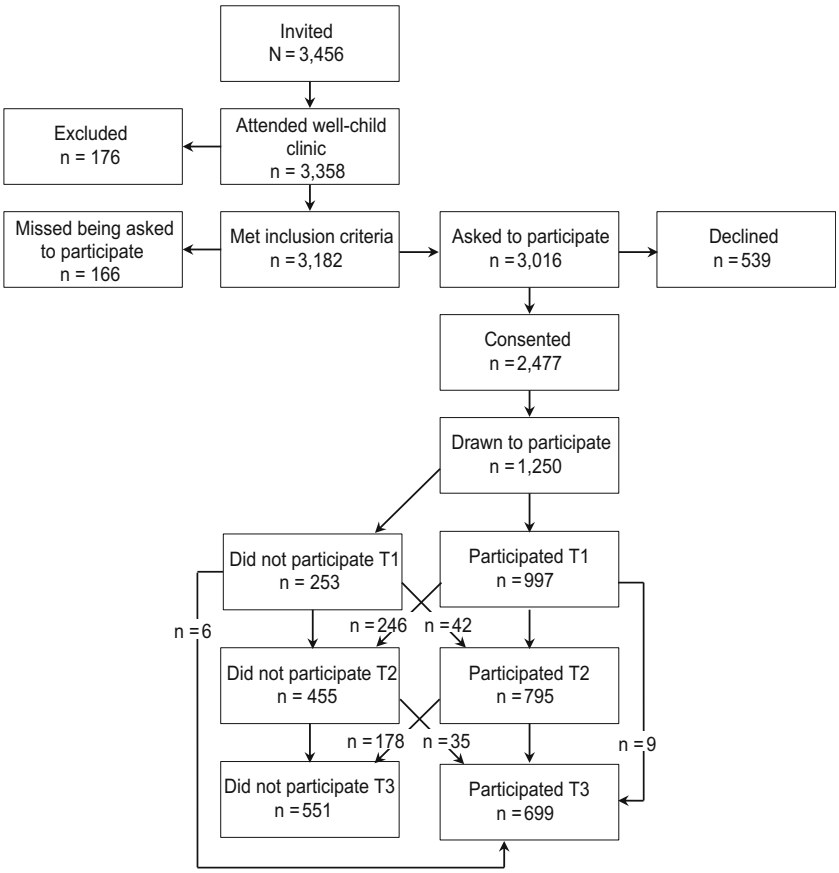

Fig. 1 Sample recruitment and follow-up. *Note*. Number of participants at the different assessment points is based on the number of participants drawn to participate ($n = 1250$), minus those who did not participate at the respective measurement point (i.e., T1, T2, T3)

Committee of Medical and Health Research Ethics, Mid-Norway approved the study (approval number 2009/994).

Missing data were handled with a Full information maximum likelihood (FIML) procedure under the assumption that data were missing at random (MAR), which implies that analyses are performed on all available data provided that cases have values for the dependent variables (n = 594). Attrition analyses revealed that children's symptoms of depression at age 4 predicted drop-out at age 8 (B = 0.32, S.E = 0.12, df = 1, $p \leq 0.01$) (but not at age 6), indicating that data were indeed MAR. Parental PD symptoms measured when children were 4 did not predict attrition at ages 6 and 8, and neither did children's symptoms of anxiety.

Measures

Parental Symptoms of Personality Disorders Given the emerging view that personality pathology is dimensional (Widiger and Simonsen 2005; Clark 2007; Trull and Durrett 2005) and because the prevalence rates of specific and subclinical PDs in community samples are relatively low (Dhawan et al. 2010; Grant et al. 2008; Grant et al. 2004), we examined the three clusters of PD symptoms distinguished in DSM-IV (American Psychiatric Association 1994) (i.e. Cluster A, B and C). Such approach is supported by the high correlation among PD's within the same cluster (Lenzenweger et al. 2007). PD symptoms were captured by the DSM-IV-related items (102 statements) of the DSM-IV and ICD-10 Personality Questionnaire (DIP-Q) (Ottosson et al. 1995). The items (e.g., "I feel very lost inside - I don't really know who I am") are shorter and more straightforward compared to the wording in the DSM but nevertheless resemble the original diagnostic criteria (Ottosson et al. 2000). Respondents are requested to indicate whether the statements are either true or false. A self-report version of the Global Assessment of Functioning (GAF) was also included in the questionnaire as well as a five-item impairment/distress (ID) scale. The ID scale corresponds to the general diagnostic criteria and aims to measure the interpersonal and major daily-life problems caused by the individuals' personality problems (5 = distress and reduced functioning; 0 = no problems) (Ottosson et al. 1998).

The DIP-Q measures dimensional scores by the number of criteria fulfilled for each individual PD, and strong dimensional agreement between structured expert clinical interviews and self-report for specific personality disorders has been found (Ottosson et al. 1998). In the present study, we used sum scores of DSM-IV-defined PD symptoms for Clusters A, B and C (i.e., continuous variables), as earlier studies have shown acceptable agreement at the cluster level (Sensitivity: Cluster A = 0.80; Cluster B = 0.75, Cluster C = 0.84; Specificity: Cluster A = 0.72; Cluster B = 0.74, Cluster C =

0.78), with intraclass correlation (ICC) estimates for the number of criteria fulfilled for Clusters A, B and C according to DIP-Q and interview reported to be 0.60, 0.78, and 0.66, respectively (Ottosson et al. 1998). To measure internal consistency we used Armor's theta, given the categorical nature of the item responses (true/false). High levels was achieved for all three clusters at both measurement points (T1/T2: Cluster A: $\theta = 0.89/0.88$; Cluster B: $\theta = 0.95/0.95$; Cluster C: $\theta = 0.89/0.90$). Finally, in the absence of comorbid disorders, a high degree of test-retest reliability has been reported for all three clusters (Ottosson et al. 2000). Of note, we also considered symptoms of specific PDs (i.e., those within a cluster).

Children's Symptoms of Emotional Disorders The Preschool Age Psychiatric Assessment (PAPA) (Egger and Angold 2004), a semi structured psychiatric interview for parents of preschool children was applied to measure children's symptoms of emotional disorders at ages 4 and 6. At age 8, both parents and children were interviewed using the Child and Adolescent Psychiatric Assessment (CAPA; Angold and Costello 2000), and a symptom was considered to be present if it was reported by either child or parent. The PAPA and CAPA interviews were administered by trained personnel with at least a bachelor's degree in a relevant field and substantial practice working with children and families. A detailed glossary provides clear definition of symptoms and the interviewer keeps asking questions until she/he can decide whether the symptoms described meet these definitions (Egger and Angold 2004), using a 3-month primary period. For the PAPA, 9% of the audio-recording were recorded by blinded raters, yielding inter-rater reliabilities of ICC = 0.91 and 0.90 for symptom counts of anxiety and depressive disorders, respectively, whereas 15% of CAPA interviews were recorded, yielding ICCs of 0.86 and 0.87.

Anxiety disorders captured by the PAPA/CAPA included separation anxiety (8 symptoms), social anxiety (2 symptoms), generalized anxiety disorder (6 symptoms), and specific phobias (7 disorders). A sum score of DSM-IV defined Major Depressive Disorder (MDD) symptoms (9 symptoms) was also calculated.

Parental Symptoms of Depression and Anxiety were treated as covariates and measured by the Beck Depression Inventory – II (Beck et al. 1988b) ($\alpha = 0.87$) and Beck Anxiety Inventory (Beck et al. 1988a) ($\alpha = 0.81$), respectively.

Analysis Plan

A fixed effects model was constructed using a structural equation modeling (SEM) framework to evaluate the prospective relations linking parental PD symptoms with children's symptoms of anxiety and depression. As illustrated in Fig. 2, in the multivariate model, autoregressive paths reflect the unique rank-order stability of parent's Cluster A, B and C symptoms and the rank-order stability of children's symptoms of anxiety and depression; cross-lagged paths capture the relations between the antecedent predictors and future outcomes: Symptoms of children's anxiety and depression at ages 6 (T2) and 8 (T3) were regressed on each of the parental PD clusters at age 4 (T1) and 6 (T2), respectively. Offspring anxiety was adjusted for parental anxiety, whereas offspring depression was adjusted for parental depression. To account for the effect of comorbidities—between childhood anxiety and depression (Melton et al. 2016), between PDs in adults (Oldham et al. 1992), and between adult PDs, anxiety and depression (Friborg et al. 2014)—within-time associations between error terms for child and parent variables were allowed. Fixed effects were added to the cross-lagged model by including a latent factor loading on children's anxiety and depression symptoms at ages 8 and 6. The latent time-invariant factor was allowed to correlate with all PD measures at ages 4 and 6 as well as children's initial symptoms of anxiety and depression at age 4. Fixed effects models have limited statistical power because they only utilize within-person variance. Random effects models, however, utilize both within- and between-person information and therefore have narrower standard errors; however, they presuppose that PDs are uncorrelated with the time-invariant latent factor – an assumption that may not be correct. To empirically test whether a fixed or random effects model fit the data best, we used Satorra-Bentler's scaled chi-square test (Satorra and Bentler 2001), which is a functional equivalent to the Hausman test (Hausman 1978). Moreover, hybrid models consisting of both fixed and random effects, in which insignificant correlations between predictors and time-invariant factor(s) are set to zero, are also possible. Such hybrid models thus retain the fixed-effects advantage while utilizing between-person information; as a result, they are more parsimonious and statistically powerful than pure fixed effects models (Allison 2009). We therefore tested whether such a model did or did not adversely affect model fit.

Sensitivity Analyses Following the same procedure as described above, we estimated three additional models (sensitivity analyses), testing (1) the prospective relation between parental dimensional sum scores for paranoid, schizotypal and schizoid PDs and children's dimensional symptoms of anxiety and depression; (2) the prospective relation between parental dimensional sum scores for antisocial, borderline, histrionic and narcissistic PDs and children's symptoms of anxiety and depression; and (3) the prospective relation between parental sum scores for avoidant, dependent and obsessive compulsive PDs and children's symptoms of anxiety and depression. Finally, because the impact of parental PDs on children's emotional disorders may change as the child grows or may be constant during the observed time-span, we tested whether fixing the effect of parental PDs to be equal over time—a

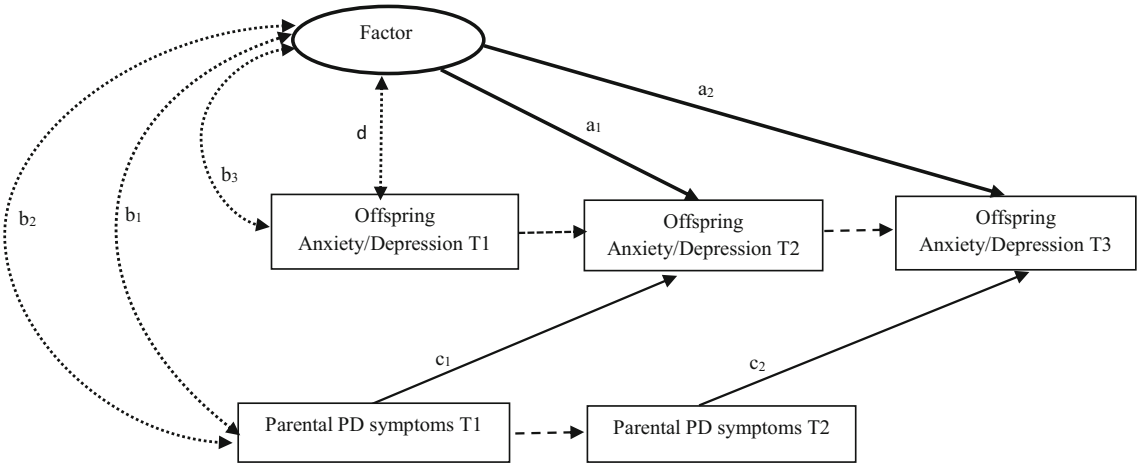

Fig. 2 Graphical representation of the hybrid fixed/random effects model of the relation between parents' symptoms of personality disorders and symptoms of anxiety and depressive disorders in their offspring. *Note*: Presentation of the analytical model tested. T1: Age 4; T2: Age 6; T3: Age 10. Note that the model is abbreviated for illustrative purposes. Within time correlations between all variables were estimated but are not displayed in the figure. Further, parental anxiety and depression were accounted for, but is not shown. The model consists of 1 time-invariant factor (latent factor), parental Cluster A, B and C symptoms (measured at T1, T2) and offspring symptoms of anxiety and depression (measured at T1, T2, T3) (Results: see Table 3). In random effects models, the correlations between parental PD symptoms (i.e., predictors) and the time-invariant factor are fixed to zero, whereas in fixed models these correlations are freely estimated. In a hybrid model, PD symptoms shown to be uncorrelated with the time-invariant factor are fixed, whereas those who are associated with the latent factor are freely estimated. Time-invariant factor part (a) and fixed (b)/random; (c) cross-lagged paths: (d) correlations. PD symptoms are allowed to correlate with each other and with offspring anxiety and depression symptoms, and symptoms of anxiety and depression are allowed to correlate with each other (not shown)

more parsimonious model—fit the data worse than freeing these parameters (see online material, M5 in Table S2).

All analyses were completed in Mplus 7.31 (Muthén and Muthén 1998-2013). To adjust for the screen-stratified sample, we weighted all analyses by weighing up´ low screen scorers and ´weighing down´ high screen scorers according to a factor calculated by dividing the number of children in the population in a specific stratum by the number of participants in that stratum. Because anxiety, depression and PDs are expected to be positively skewed, a Robust maximum likelihood estimator, which does not presuppose multivariate normality, was deployed. This estimator also provides robust standard errors.

Results

Preliminary Analyses

Means and intercorrelations of all study variables at baseline are displayed in Table 1.

As expected, all PD cluster symptoms were moderately to strongly positively correlated with each other. There was also a moderate positive correlation between children's symptoms of anxiety and depression.

Primary Analyses

The results of the model fitting procedure are depicted in Table 2. A fixed effects model (M3) did fit the data better than a random effects model (M2). Inspection of M3 results indicated that symptoms of parental PDs did not correlate significantly with the time-invariant latent factor and setting these correlations to zero (M4) did not adversely affect model fit. Such hybrid models are more parsimonious and more statistically powerful than purely fixed effects models (Firebaugh et al. 2013; Allison 2009); thus, model M4 was preferred. The parameter estimates of the effect of PDs on anxiety and depression in M4 are portrayed in Table 3. As seen, more parental Cluster C symptoms when children were 4 and 6 years of age forecasted more symptoms of anxiety in children two years later, whereas the path from parental Cluster A symptoms to offspring anxiety was evident from age 6 to 8 exclusively. A Wald test of parameter constraint (i.e., constraining the paths from age 4 to 6 and from age 6 to 8 to be equal) revealed that the effect of parental Cluster A symptoms on offspring anxiety from age 6 to 8 was indeed stronger than that from age 4 to 6 (Wald $\chi 2 = 4.79$, $df = 1$, $p = 0.029$). As further displayed in Table 3, elevated levels of parental Cluster B symptoms when children were 6 years forecasted more symptoms of depression in offspring two years later. Recall that these results emerged after taking into account all time-invariant unmeasured confounders (e.g., genetics), baseline levels of children's anxiety and depression, and parental anxiety and depression. Of note, parental PDs were highly stable over this two-year span (range: $\beta = 0.60$–0.64).

To investigate whether the seemingly linear effect of Cluster A, C and B symptoms on children's symptoms of emotional disorders were truly linear or whether the effect

Table 1 Means, standard deviations and bivariate correlation coefficients between all study variables ($n = 594$)

Psychiatric symptoms (possible range)	Mean (SD)	1	2	3	4	5	6	7	8	9	10	11	12
1. P Cluster A T1 (0–35)	0.91 (1.00)	1	0.68***	0.49***	0.41***	0.41***	0.39***	0.03	0.12**	0.06	0.17***	0.08*	0.10*
2. P Cluster A T2 (0–35)	0.89 (1.00)		1	0.42***	0.46***	0.40***	0.51***	0.03	0.15***	0.08	0.12**	0.07	0.11***
3. P Cluster B T1 (0–46)	0.85 (0.78)			1	0.70***	0.58***	0.43***	0.09**	0.08	0.14**	0.19***	0.12**	0.15***
4. P Cluster B T2 (0–46)	0.80 (0.76)				1	0.39***	0.49***	0.08	0.14**	0.14**	0.18***	0.17***	0.17***
5. P Cluster C T1 (0–25)	1.53 (0.99)					1	0.68***	0.05	0.15***	0.15***	0.19***	0.17***	0.14***
6. P Cluster C T2 (0–25)	1.46 (1.00)						1	0.13***	0.19***	0.19***	0.20***	0.17***	0.18***
7. C Anxiety T1 (0–23)	0.69 (1.20)							1	0.13***	0.27***	0.36***	0.13**	0.16**
8. C Anxiety T2 (0–23)	0.86 (1.5)								1	0.28***	0.16**	0.43***	0.21***
9. C Anxiety T3 (0–23)	0.89 (1.2)									1	0.21***	0.25***	0.45***
10. C Depression T1 (0–9)	0.45 (0.74)										1	0.23***	0.25***
11. C Depression T2 (0–9)	0.52 (0.86)											1	0.32***
12. C Depression T3 (0–9)	0.48 (0.79)												1

P, Parent; C, Child. *$p \leq 0.05$. **$p \leq 0.01$. *** $p \leq 0.001$

was stronger at one or both ends of the PD continuum, quadratic components of Cluster A, C and B symptoms were added to the model (M4) while mean-centering the PD variables. None of these quadratic components predicted offspring anxiety and depression (all $ps > 0.09$), thereby indicating that the transgenerational relationship between symptoms of PDs and emotional disorders were not specific to either the low or high end of the PD spectrum.

Sensitivity Analyses

Our sensitivity analyses (see online material, Table S1 and Table S2) using symptoms of specific PDs within each cluster (e.g., Cluster A: sum scores of paranoid PD symptoms, schizotypal PD symptoms and schizoid OD symptoms, respectively, vs. a sum score of symptoms for all Cluster A PDs (main analysis)) as the predictor revealed the following results: (1) Cluster A: Children's anxiety was only predicted by parent's schizotypal PD symptoms, but neither schizotypal, paranoid nor schizoid sum scores of symptoms were related to offspring depression, consistent with the results from the primary analyses; (2) Cluster B: Children with parents who displayed higher levels of borderline PD symptoms had increased risk of depressive symptoms two years later, adjusting for baseline levels of depression, parental depression and the remaining Cluster B PD symptom sum scores (antisocial, histrionic, narcissistic) (Tables S1 and S2); (3) Cluster C: Higher levels of anxiety symptoms in children were evident if parents displayed more symptoms of obsessive compulsive PD symptoms two years earlier. Children's symptoms of depression were specifically predicted by parents' dependent PD symptoms (Tables S1 and S2). In sum, the results of the sensitivity analyses were in accordance with the main results.

Discussion

Although it has been theorized that parental PDs may increase the risk of emotional disorders in offspring, this possibility has never been examined prospectively. We therefore evaluated whether parental PD symptoms corresponding to clusters A, B and C, measured when children were 4 and 6 years of age, predicted children's symptoms of anxiety and depression two years later, net of prior child symptoms, parental anxiety and depression, and all unmeasured time-invariant confounding. Consistent with expectations, more parental Cluster A and C symptoms predicted more symptoms of anxiety in offspring two years later, whereas elevated levels of parental cluster B symptoms forecasted more symptoms of depression.

It should be noted that the level of the sum of symptoms going into Clusters A, B and C was rather low (averaging 0.9, 0.8, and 1.5, respectively), whereas substantially more symptoms—ranging from 3 to 5—are required to meet criteria for a specific Cluster A, B or C diagnosis (American

Table 2 Results of model fitting procedure – Cluster A, B, C symptoms (n = 594)

	X^2	df	*P* value	ΔX^2	df	P value	RMSEA[a] (90% CI)	SRMR[b]	CFI[c]	TLI[d]
M1: Baseline model	1348.22	84	<001							
M2: Random effects	77.03	30	<001				0.051 (0.037, 0.066)	0.040	0.963	0.896
M3: Fixed effects	43.01	22	0.005	42.47	8	<001	0.040 (0.022, 0.058)	0.022	0.983	0.937
M4: Hybrid model with random and fixed effects	51.12	28	0.005	4.51	6	0.61	0.037 (0.020, 0.053)	0.024	0.982	0.945

$\Delta\chi^2$ is corrected according to Satorra-Bentler's procedure. Preferred model in bold. [a] Root mean square error of approximation; [b] Standardized root mean square residual; [c] Comparative fit index; [d] Tucker Lewis Index. NA – Not applicable

Psychiatric Association 2013). Thus, the prevalence of formal PD diagnoses in the clusters was in all likelihood low in our community sample. Importantly, there were no nonlinear effects of parental PD symptomatology, indicating that the results were not driven by those parents who were very high (or low) on PD symptomatology. Thus, even low levels of Cluster A, B and C symptoms in parents would seem to contribute to children's symptoms of emotional disorders, given the dose-response relation we documented. Further, even though the association under consideration was relatively modest in magnitude, its theoretical and practical significance should not be discounted (McCartney and Rosenthal 2000). Because the effects of PD symptoms may accumulate over time, the total influence of parents' PD symptoms on emotional problems in offspring may be underestimated when only a short period of development is the focus of inquiry (Prinzie et al. 2009). Indeed, evidence indicates that the relation between personality and parenting, the latter potentially mediating the revealed association between parental PDs and offspring symptomatology, gets stronger over time (i.e., stronger in longitudinal than in cross-sectional studies) (Prinzie et al. 2009).

Cluster A and C Symptoms Predict Anxiety Symptoms in Offspring

Findings linking Cluster A and C symptoms with increased levels of anxiety symptoms in children are consistent with Rutter and Quinton's research (1984) showing that children of parents with PDs are at increased risk for global emotional disturbance. The current inquiry extends this early work by revealing a distinctive relation between parental Cluster A and C symptoms and anxiety symptoms in offspring, net of time-invariant factors.

Cluster C is defined as the "anxious-fearful" cluster and is highly comorbid with anxiety disorders, as is Cluster A (Friborg et al. 2013). However, because we accounted for parental anxiety and depression, the results do not simply reflect the association between parental and child anxiety. This fact, together with our reliance on a hybrid fixed effects model in which the influence of all unmeasured time-invariant confounders were ruled out (Firebaugh et al. 2013; Allison 2009), indicates that the results reported herein reflect the specific effect of Cluster A and C symptoms over and above direct genetic or genetically mediated effects. Of note, though, the path between parental Cluster A symptoms and offspring anxiety symptoms was evident from age 6 to 8 only, and it significantly differed from the age-4-to-6 path. This indicates that the impact of parental Cluster A symptoms on offspring anxiety is age-dependent. Although this study needs to be replicated, our finding can be seen in the light of a former community study showing that parents with Cluster A PD symptoms make more frequent attempts to influence or control their children than other parents (Wilson and Durbin 2012). Such control might have a more negative, profound impact on offspring with increasing age and autonomy. Note as well that parents with higher levels of neuroticism (i.e., anxious, tense, or more easily distressed) tend to provide their children less autonomy support (corresponding to overprotection and control) than other parents (Prinzie et al. 2009). Further, Cluster-C parents are also disproportionately likely to engage in more controlling parenting (Wilson and Durbin 2012). Consider as well evidence showing that increases in negative mother-child relationship quality and decreases in positive relationship quality are accompanied by an escalation of internalizing problems in offspring (Brock and Kochanska 2015). In light of these observations from other studies and the results discerned in the current inquiry, future research should test whether the Cluster A- and C-specific effects are mediated by problematical parenting as well as examine potential age-dependent effects.

Cluster B Symptoms Predict Depression Symptoms in Offspring

Our hypothesis stipulating that Cluster B symptoms would be specifically related to depressive symptoms in offspring was also confirmed, and our sensitivity analyses revealed that there was a specific path between borderline PD symptoms (Cluster B) and offspring depression. These findings are in good accordance with a recent review (Eyden et al. 2016) indicating that children of mothers with borderline personality disorder are at heightened risk of depression, even if maternal BPD pathology has more often been linked to externalizing problems in offspring. In any event, the depression-specific

Table 3 Children's symptoms of anxiety and depressive disorders at ages 6 and 8 years predicted from parents' symptoms of personality disorders two years earlier, adjusted for baseline levels of children's symptoms and parental symptoms of anxiety and depression, respectively*

	Children's symptoms of anxiety disorders								Children's symptoms of depressive disorders							
	Age 6				Age 8				Age 6				Age 8			
Predictors: Parents' symptoms of personality disorders two years earlier	*B*	95% CI	*β*	*p*	*B*	95% CI	*β*	*p*	*B*	95% CI	*β*	*p*	*B*	95% CI	*β*	*p*
Cluster A	0.15	0.01, 0.29	0.10	0.033	−0.05	−0.17, 0.07	−0.04	0.450	−0.01	−0.09, 0.07	−0.01	0.881	0.01	−0.08, 0.09	0.01	0.899
Cluster B	−0.06	−0.23, 0.11	−0.03	0.472	0.09	−0.07, 0.25	0.06	0.289	0.03	−0.08, 0.14	0.03	0.600	0.11	0.01, 0.22	0.11	0.04
Cluster C	0.19	0.06, 0.33	0.13	0.005	0.15	0.03, 0.28	0.13	0.018	0.09	−0.00, 0.19	0.11	0.056	0.09	−0.01, 0.18	0.11	0.068
Covariates: Parents' symptoms of anxiety/depression two years earlier																
BAI	−0.20	−0.07, 0.03	0.04	0.425	0.02	−0.03, 0.06	0.05	0.464	a	a	a	a	a	a	a	a
BDI	a	a	A	a	a	a	a	a	0.02	−0.00, 0.04	0.09	0.113	0.00	−0.01, 0.02	0.03	0.656

* Hybrid fixed and random effects model (Model 4, Table 2) (N = 594)

BAI, Beck Anxiety Inventory; BDI, Beck Depression Inventory –II; *B*, unstandardized beta coefficients; *β*, standardized beta coefficients. a, not estimated (only parent's anxiety symptoms were accounted for when the outcome was children's anxiety symptoms, whereas parental depression symptoms were accounted for when the outcome was children's depressive symptoms)

findings that we observed extend the results of earlier studies by showing that Cluster B symptoms (i) forecast depressive symptoms in (ii) a community sample of children, using (iii) a structured psychiatric interview (i.e., not questionnaires), (iv) after accounting for parental depression and (v) applying a statistical method that adjusts for all unmeasured time-invariant confounders.

Future research should seek to illuminate whether the B-cluster results just summarized are mediated by parenting, marital conflict and/or other potential mechanisms. Consistent with the parenting possibility is evidence that BPD mothers, relative to other mothers, evince more disruptive affective communication with their infants (Hobson et al. 2009), which increases the risk for disorganized attachment (Madigan et al. 2006) and, thus, the likelihood for later depression (Morley and Moran 2011). Childhood depression is also related to parental rejection and low emotional warmth (Beesdo et al. 2010; McLeod et al. 2007a), and although findings are somewhat inconsistent, BPD mothers (Cluster B) display less maternal warmth and more rejection than other mothers in addition to being more negative and hostile (Eyden et al. 2016). Of note, although time-invariant confounders were accounted for, unmeasured time-*variant* factors may still act as confounders and explain the present findings. For example, stressful life-events affecting both parents and children (e.g., loss of a dear one, marital break-up, severe accidents or prolonged hospitalization of family members) may aggravate PDs and impair parental functioning (Pagano et al. 2004) as well as increase the risk of offspring psychopathology (Williamson et al. 2005). Such potential time-varying confounders should be accounted for in future work.

Clinical Implications

Although our results are in need of replication, our findings first indicate that parental PD symptomatology should be considered as a relevant psychopathogenic vector when children present with anxiety or depression. Crucially, disturbances in children should not be presumed to be simply the result of genes shared with their parents given the analytic approach employed herein, which discounted such genetic mediation. To the extent that parental PD symptoms can themselves be reduced, there is reason to believe that children will benefit from interventions targeting caregivers and parenting styles. Although interventions specifically designed for parents with PDs and their children have not yet been developed, attachment-based interventions (e.g., Circle of Security) (Hoffman et al. 2006) and/or psychoeducation-based interventions for parents with mental health problems are promising (Cohen et al. 2008) and may therefore inform interventions addressing parents with PD symptomatology and their offspring.

Limitations

The results of this study should be interpreted in the context of its limitations. First, in our analyses, both PDs and anxiety/depression were analyzed as symptom counts, and even though there is not convincing evidence for disorders being categorical in nature (Haslam et al. 2012), our findings do not necessarily generalize to diagnoses of PDs and emotional disorders. Nevertheless, it seems noteworthy that even though the mean count of symptoms or diagnostic criteria were low in this sample, they still seemed to affect the children. Second, because the DSM-IV does not weigh the symptoms within each disorder, they are also not weighed in the PAPA/CAPA, and thus not in our anxiety measure. Of note, apart from social anxiety, the number of symptoms/disorders is fairly equally distributed between the anxiety disorders. Thus, our anxiety measure does not favor social anxiety, and the results should be interpreted with this in mind. Third, although the influence of time-invariant factors (e.g., trait-like reporting bias, genetics) was ruled out, uncontrolled time-varying factors may still be responsible for increases (or decreases) in parents' PD symptoms and, thus, children's anxiety and depression. Fourth, parents reported on both their own and their children's symptoms. Although such a methodological feature is typically a cause for concern, recall that the statistical approach obviated this problem by accounting for all time-invariant unmeasured confounders (Firebaugh et al. 2013). Finally, given the proof-of-concept conceptualization of this study (i.e., proving the transgenerational impact of parental PD on offspring's early developmental emotional disorders), we used canonical DSM-IV PD designations (with related clusters and dimensional scores) and abstained from more sophisticated formalizations of PD traits (e.g., bifactorial models). This approach might have obscured subtle intergenerational effects of other components. Likewise, we limited the analysis to rather broad psychopathological symptoms (i.e., depression and anxiety) without considering more severe symptoms.

Summary and Conclusion

The results of our community study suggest that parental symptoms of Clusters A and C (particularly schizotypal PD and obsessive-compulsive PD) increase future symptoms of anxiety in children, whereas elevated levels of Cluster B (particularly borderline PD) symptoms in parents seem to increase symptoms of depression in offspring. Given that initial symptoms of anxiety and depression, parental anxiety and depression and all unmeasured time-invariant potential confounders were accounted for, we regard the selectivity of the transgenerational effect (i.e., the effect of parental PD traits on the emotional disorders of their offspring in early childhood) as highly relevant. Clinically, this implies that, when children present with anxiety or depression, parental PD symptomatology, even at subclinical trait levels, is important to consider. Moreover, when parents are the initial target of mental health services, the emotional problems of their offspring should be considered, as should the prospect that successful treatment of the parents' Cluster A, B and C traits might benefit their children. Future studies should aim to capture the potential mechanisms explaining the present findings, including the mediating role of parenting.

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Compliance with Ethical Standards

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