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Personality disorders in offspring of mothers with mood disorders: Results from a longitudinal family study

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ABSTRACT

Offspring of mothers with mood disorders are known to be at risk for a range of adverse outcomes, but the prevalence of personality disorders (PDs) in this group is unknown. The goal of this study was to assess risk of PD diagnoses and symptoms in offspring of mothers with and without mood disorders, and to explore contributing factors to this risk. This longitudinal study assessed PDs and symptoms of PDs in offspring of mothers with bipolar disorder (O-BD), major depression (O-MDD), and no psychiatric diagnosis (O-WELL) in mid-adolescence and in early adulthood. O-BD were more likely to develop a Cluster B PD than O-MDD or O-WELL in adolescence, and more likely to develop a Cluster B PD then O-WELL in early adulthood. Dimensional analyses revealed that O-BD had elevated symptoms in PDs across all PD clusters at mid-adolescence and young adulthood. O-MDD showed elevated symptoms of antisocial PD at both time points, and of obsessive-compulsive PD at young adulthood. Offspring of mothers with mood disorders, especially O-BD, are at increased risk for PD diagnoses and symptoms in mid-adolescence and early adulthood. Contributing factors to risk of PD symptoms in at-risk offspring are discussed.

1. Introduction

Mood disorders, such as bipolar disorder and major depressive disorder (MDD), represent a serious health problem for individuals, their families and society. Offspring of depressed mothers provide a group in which both rearing and biological risk factors are present, substantially increasing the risk for psychopathology. Studies of offspring of parents with bipolar disorder (O-BD) and offspring of parents with major depressive disorder (O-MDD) have demonstrated elevated risk for a broad range of problems, including a higher incidence of bipolar disorder and MDD in comparison to offspring of well parents (O-WELL), a higher incidence of other psychiatric disorders, and functional impairment including poor social and academic functioning (Birmaher et al., 2009; Bruder-Costello et al., 2007; Egeland et al., 2012; Mesman et al., 2013; Rasic et al., 2014; Weissman et al., 2006; Zahn-Waxler et al., 1988). Elevated risk for psychopathology in offspring of parents with mood disorders could include personality disorders (PDs). High rates of co-morbidity between mood disorders and PDs are often reported (Brieger et al., 2003) and patients with these conditions often have overlapping family histories (Akiskal et al., 1985b; Weller et al., 1994) and mood disorders (Bienvenu et al., 2011) are heritable. However, the prevalence of PDs in offspring of parents with mood disorder has not been fully examined.

Several cross-sectional studies have identified patterns in offspring of parents with mood disorders on constructs relevant to personality development. Offspring of parents with mood disorders are often characterized by difficult temperaments (reviewed by Chang et al. (2003)). In comparison to low-risk children, O-BD have exhibited behavioral disinhibition, including hyperthymic personality, novelty-seeking, and extroversion (Hirshfeld-Becker et al., 2003); greater dysregulation as measured by the Child Behavior Checklist (CBCL) (Diler et al., 2011); and increased activity levels and decreased task orientation (Singh et al., 2008). Additionally, a difficult temperament is more likely in O-MDD compared to O-WELL (Bruder-Costello et al., 2007).

Research has also demonstrated strong links between temperamental risk factors in offspring and offspring psychopathology. In recent reports from a longitudinal study of O-BD, high emotionality predicted psychopathology and mood disorder in O-BD (Doucette et al., 2013) and both high emotionality and shyness

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predicted the development of anxiety disorders in O-BD, which subsequently increased the risk of mood disorders (Duffy et al., 2013). Another study suggested that certain offspring personality traits (neuroticism, extraversion and psychoticism) are associated with offspring mood disorders but not with parent mood disorders (Rothen et al., 2009). However, other research suggests that among parents with mood disorders, parent personality traits could play a role in the development of their offspring. Research examining O-BD and O-WELL showed that high levels of parental neuroticism and low agreeableness predicted poor interpersonal functioning of the offspring during late adolescence-early adulthood, and this relationship was especially strong among the O-BD (Ostiguy et al., 2012). Overall, these studies indicate that offspring of parents with mood disorders develop problematic personality traits, which could be early characteristics of PD psychopathology.

Longitudinal work is promising for explaining how personality functioning unfolds across development. Earlier results from the present longitudinal study showed that in childhood, O-BD had heightened distress and preoccupation with conflicts, difficulty maintaining friendly social interactions, and trouble modulating hostile impulses (Zahn-Waxler et al., 1984). Later work on this sample suggested differential patterns for how problems unfold over time, such that for O-MDD, early self-regulatory deficits cascade into internalizing problems, but these early deficits cascade into thought problems for O-BD (Klimes-Dougan et al., 2010). However, no longitudinal studies have yet specifically examined how maternal mood disorder diagnosis impacts the risk of PDs in these offspring.

The goal of the present study was to measure PD psychopathology in O-BD, O-MDD, and O-WELL. In this longitudinal study, we examined PDs at two assessment points, in late adolescence and in early adulthood. Considering the possibility of low base rates for full-threshold PD outcomes in the offspring, as well as the increasing emphasis on the importance of dimensional approaches in PD research (Krueger, 2013), we used both categorical and dimensional approaches in our analyses. For the categorical approach, we aggregated PDs across PD clusters A, B and C; for the dimensional analyses, we examined symptom levels of all 10 DSM-IV PDs. We predicted that offspring of mothers with mood disorders would have greater PD psychopathology than O-WELL at both time points. In particular, based on prior work showing the most severe developmental deviance by adolescence and young adulthood (Klimes-Dougan et al., 2010) in O-BD, we predicted that this group would show the most PD psychopathology. We further assessed whether maternal mood disorder diagnosis would predict PD outcomes over and above the impact of a range of other relevant factors such as maternal PD, maternal substance use disorders, maternal global assessment of functioning (GAF), family stress, and the presence of a mood disorder in the offspring. A secondary goal was to assess within-individual continuity of PD symptoms across the T4 and T5 assessments. We predicted moderate levels of continuity in PD symptoms across time.

2. Methods

2.1. Sample

This study is based on archival data from a longitudinal investigation of O-BD, O-MDD and O-WELL. Recruitment and ongoing assessments took place between 1979 and 2003. All mothers were the biological mothers and the primary caregivers for the offspring. Additional study details can be found in the previous publications from this project (Zahn-Waxler et al., 1988). The families in this study were seen five times during the offspring's development, starting from early childhood extending through young adulthood. The first four assessments were about 3 years apart (T1, T2, T3, and T4) and the final assessment was about 7 years later (T5). During these five visits, comprehensive assessments were conducted on parents' and children's psychiatric status, children's psychosocial functioning, and families' functioning. Here we report on the subset of offspring who completed personality assessment at either or both of the T4 (mid-adolescence) and T5 (early adulthood) assessments.

Of the 126 families meeting the initial criteria for participation in the longitudinal study, 114 families were considered eligible for this study at the time of the T3 assessments (e.g., families whose mother retained a diagnosis of minor depression were initially included in the recruitment efforts but ruled out as eligible for participation after T3). Of these, 98 eligible families participated through T3, and 91 families participated in the T4 and/or the T5 visit. Based on the initial sample, families with lower Socioeconomic scale (SES) and with male young adult offspring had greater attrition. Included in this study were 146 offspring participants at T4 and 136 offspring participants at T5; 115 offspring participated in both the T4 and T5 assessments.

2.2. Parental diagnostic assessment

At recruitment (T1), mothers were administered the Schedule for Affective Disorders and Schizophrenia: Lifetime Version (SADS-L) (Spitzer et al., 1978). The interviews were conducted by a psychiatric nurse who had been trained by a staff member of the New York Psychiatric Institute ($\kappa = 1.0$). Families were eligible if mothers met the Research Diagnostic Criteria for bipolar disorder (I or II) or MDD or if they were without past or current psychiatric disorder; their offspring were correspondingly grouped to O-BD, O-MDD and O-WELL. If the mother was eligible, the SADS-L interview was also administered to the father. The number and percentages of fathers that had at least one psychiatric diagnosis are summarized across offspring groups in Table 1. For the well families, both parents had to be without current or past psychiatric disorders. From the initial diagnostic interview, clinicians rated the mothers on the global assessment scale (GAS) (Endicott et al., 1976). The average GAS score for the depressed mothers was 43.22 (S.D. = 19.77) at a level of "serious symptomatology or impairment in functioning that most clinicians would think obviously requires treatment or attention" (Endicott et al., 1976, p. 176).

Six years into the study, mothers were re-diagnosed using the Structured Clinical Interview for DSM-III-R (SCID) (Spitzer et al., 1990) and the Interval SADS. Nine mothers' diagnoses were changed (seven of which changed in the type of depression manifest from minor depression to MDD or from MDD to bipolar disorder). The diagnosis used in this study is the mother's adjusted "lifetime" diagnosis. Fathers were also re-diagnosed using the Interval SADS. As shown in Table 1, two-thirds of the fathers of the O-MDD adolescents had at least one psychiatric disorder (mood disorder, anxiety disorder, and/or substance abuse disorder). Changes in offspring grouping were based exclusively on changes in maternal diagnosis. Finally, information about stress in the family, including health problems, family conflicts, financial issues, loss of significant people, and marital discord, was collected using the Brown–Harris schedule for assessing family function (Brown and Harris, 1978).

Mothers' personality assessment was conducted at T3 using the Personality disorders examination Personality disorders examination (PDE) (Loranger, 1988). This is a semi-structured clinical interview for diagnosing PDs consisting of 126 questions assessing DSM-II-R criteria rated on a 3-point scale. The same clinician who administered the SADS at T3 administered the PDE. This measure has been compared with the SCID-II and with consensus diagnosis and has shown moderate agreement using categorical measures and strong agreement using dimensional measures (Spitzer, 1983). Reliability for the PDE in this study was based on a second clinician rating for 20% of the cases that were audio recorded. The results yielded an average interclass correlation of 0.90 for the individual disorder dimensional scores.

2.3. Offspring PD assessment as adolescents (T4) and young adults (T5)

We used different tools to identify PDs in adolescence (T4) and early adulthood (T5) in order to ensure that the assessments were developmentally appropriate. At T4, the Schedule for Nonadaptive and Adaptive Personality-Youth Version (SNAP-Y) was administered. The SNAP-Y (Clark, 1993) was originally developed to assess psychopathology in terms of the underlying trait dimensions that span normal through pathological personality characteristics; it also has scales to assess PD criteria from the Diagnostic and Statistical Manual (DSM-III-R) (Melley et al., 2002). The Youth Version is a 375-item self-report instrument. For each item, respondents decide how well it describes them and mark "true" if the statement is true or mostly true for them and "false" if it is false or mostly false for them. The three higher-order core personality traits and the 12 lower-order personality dimensions were derived by factor analysis. Normative data are based on a sample of 381 adolescents ages 12–18 years. The instrument demonstrates good structural and external validity as well as retest reliability (Linde et al., 2013). The scales used in the present work are derived from the existing DSM-III-R criteria, and this assessment included categorical assignments ("diagnosis present") as well as *T*-scores for symptom levels for the following PDs: paranoid, schizoid and schizotypal (Cluster A); antisocial, borderline, histrionic and narcissistic (Cluster B); avoidant, dependent and obsessive-compulsive (Cluster C).

At T5, offspring personality psychopathology was based on the Structured Clinical Interview for DSM-IV Axis II Disorders (SCID-II) (First et al., 1995). Trained clinicians, including a psychiatrist and two advanced doctoral clinical psychology students, administered the SCID-II. Clinicians were blind to maternal Axis I and Axis II diagnoses. Based on standard evaluation procedures, the order of assessment of disorders was designed to maximally facilitate rapport with the client, such that Cluster A disorders were assessed last. For this assessment, categorical assignments were again determined, as well as dimensional measures (symptom counts) for the DSM-IV 10 PDs.

2.4. Assessment of Axis I psychopathology in offspring during adolescence and adulthood

The adolescent (T4) assessment included screening for the presence of current or past mood disorders using the mood disorder sections of the Diagnostic Interview for Children and Adolescents-Revised (DICA-R), a clinician-administered interview (Herjanic and Reich, 1982). The DICA-R was administered by masters or doctoral level trained clinicians. Reliability evaluation of results in 26 of the cases revealed an interclass correlation for affective symptoms of 0.91. Offspring received a combined diagnosis, based on both mother and child reports.

At T5, offspring Axis I disorder diagnosis was based on the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I) (First et al., 1996). Trained clinicians, including a psychiatrist and two advanced doctoral clinical psychology students, administered the SCID-I. Clinicians were blind to maternal Axis I and II diagnoses.

2.5. Statistical analysis

Statistical analyses were conducted in SAS Version 9.2 (SAS Institute, Inc., Cary, NC). The main question of the present study was to examine whether there were differences in offspring personality outcomes between the maternal lifetime diagnosis groups of O-BD, O-MDD and O-WELL. Personality outcomes based on the 10 PDs, or the 3 PD clusters, were examined using categorical outcomes, defined by the SNAP-Y at T4 or the SCID-II at T5 for a given PD based on the assessment used. For the categorical analyses, we used generalized estimating equations (GEE) for logistic regression to examine the effect of maternal risk group on meeting criteria for a given PD diagnosis, taking into account the clustering effect of multiple siblings within families.

For the dimensional analyses, we used GEE for linear regression to examine the effect of maternal risk group on PD symptom levels, again taking into account the sibling clustering effect. A series of analyses was used to evaluate symptom levels for each PD at T4, as assessed by the SNAP-Y. A second series of analyses was conducted to evaluate symptom levels for each PD at T5, as assessed by the SCID-II.

Although the primary analyses lacked power to include numerous offspring and family characteristics because of the modest sample size, follow-up analyses were conducted for all models that yielded significant differences by maternal lifetime diagnosis group to ensure that the impact of maternal mood disorder diagnosis on offspring PD outcomes was not better explained by other potential factors. For these analyses, we included as covariates the demographic as well as offspring and family characteristics that were significantly different between offspring groups: SES, maternal global assessment of functioning (GAF), and family stress (using an average of all 5 Brown–Harris stress measures). Additionally, we included maternal PD diagnosis (zero, one or multiple maternal PDs), maternal substance use disorders (any substance use disorder versus no substance use disorders) and offspring mood disorder diagnosis (any mood disorder versus no mood disorder at the time point of the PD assessment) as these have potential importance in the emergence of offspring PD psychopathology.

Finally, to examine continuity of PD symptoms across the two time points, we examined correlations between T4 and T5 symptom levels for all offspring who attended both visits. Partial correlation analyses, correcting for SES, were conducted with the whole sample together.

3. Results

3.1. Demographic and clinical characteristics of the families

Demographic characteristics of the sample are summarized in Table 1. There were a total of 167 offspring who were the focus of this study, comprised of 42 O-BD, 73 O-MDD, and 52 O-WELL, from 91 families (maximum number of siblings per family=2), who participated at T1, at T3, and at T4 and/or T5 and had also completed a PD assessment. Offspring participants were 54.8% female at T4 and 58.1% female at T5. Mean age at T4 was 15.2 years (S.D.=2.6), and at T5 was 22.4 years (S.D.=3.7). At T1, families were predominantly middle class to upper-middle class; the Average Socioeconomic Scale (SES) (Hollingshead, 1975) score was 51.9 (S.D.=14.5). There were no group differences for sex or age at T4 or at T5, but there were differences in SES at T1, where

Table 1
Demographic and clinical characteristics for offspring.

Group	Total sample	Well offspring	Bipolar offspring	Major depression offspring	Group difference statistics[a]
Total N	167	52	42	73	–
N at T4	146	44	37	65	–
N at T5	136	41	35	60	–
Sex (% female) at T4	54.8	52.3	64.9	50.8	0.38
Sex (% female) at T5	58.1	56.1	62.9	56.7	0.82
Race (% non-White) at T4	12.3	0.0	18.5	16.2	0.003
Race (% non-White) at T5	11.8	7.3	11.4	15.0	0.59
Age (years) at T4 [mean (S.D.)]	15.2 (2.6)	15.1 (2.7)	14.6 (2.7)	15.7 (2.6)	0.14
Age (years) at T5 [mean (S.D.)]	22.4 (3.7)	22.8 (5.5)	21.3 (2.3)	22.8 (2.5)	0.12
SES at T1 [mean (S.D.)]	51.9 (14.5)	58.0 (8.3)	50.7 (14.6)	48.3 (16.7)	0.0008
Stress measures mean (S.D.)					
(1) Financial		2.4 (2.2)	5.1 (2.8)	5.5 (3.2)	<0.0001
(2) Health		6.5 (2.7)	9.0 (2.1)	8.4 (2.0)	0.0006
(3) Loss		2.7 (2.2)	5.2 (2.4)	4.6 (2.6)	0.001
(4) Family		1.9 (1.8)	6.0 (3.2)	5.3 (3.2)	<0.0001
(5) Marital		3.0 (2.1)	8.7 (3.2)	7.5 (2.7)	<0.0001
Mother GAF at T3		79.5 (8.5)	56.6 (13.4)	64.7 (10.5)	<0.0001
N (%) maternal anxiety disorders	16 (17.6)	2 (7.4)	7 (29.2)	7 (17.5)	0.13
N (%) maternal substance disorders	19 (20.9)	1 (1.1)	12 (50.0)	6 (15.0)	<0.0001
Number of maternal MDD episodes [mean (S.D.)]		0 (0)	3.6 (2.0)	2.7 (2.2)	0.15
N (%) Presence of psychiatric disorders in the fathers	41 (47)	1 (4)	15 (65)	25 (66)	<0.0001
Offspring bipolar disorder at T4 [number (%)]	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Offspring bipolar disorder at T5 [number (percent)]	8 (5.8)	0 (0)	5 (13.9)	3 (4.9)	0.02
Offspring depressive disorder at T4[b] [number (percent)]	37 (22)	8 (15.4)	10 (23.8)	19 (26.0)	0.36
Offspring MDD at T5 [number (%)]	34 (24.6)	10 (27.8)	16 (26.2)	8 (19.5)	0.68
Offspring anxiety disorders at T5	34 (25.0)	7 (17.1)	13 (35.1)	14 (23.0)	0.36
Substance use disorder T5 [number (%)]	43 (31.2)	11 (26.8)	13 (35.1)	19 (31.1)	0.69

SES=Socioeconomic status; S.D.=standard deviation; GAF=Global Assessment of Function; and MDD=Major depressive disorder.

[a] Two-sided *p*-values from one-way ANOVA (age, SES) or Fisher's exact test (all others).

[b] Offspring depressive disorders at T4 included MDD and dysthymia.

the WELL families had the highest SES, and the MDD families had the lowest (F (2)=7.5, p=0.0008). Additionally, as shown in Table 1, BD families had the greatest levels of stress across all five measures in the Brown–Harris assessment of family functioning (health problems, family conflicts, financial issues, loss of significant people, and marital discord). Maternal GAF levels were lowest for those with BD and highest for WELL mothers. The presence of a maternal substance use disorder was most common in the BD families. However, there were no significant group differences with respect to the presence of maternal anxiety disorders. There was not a significant difference in number of prior MDD episodes for the BD versus the MDD families. Finally, at T4, O-MDD had the largest percentage of non-white participants (Fisher's exact test p value=0.003) but this difference was not present among participants that completed the T5 evaluation (Fisher's exact test p value=0.59.)

3.2. Prevalence of personality disorder across offspring

The prevalence rates for individual PD diagnoses in the offspring overall were relatively low (range from 0% to 30%). Table 2 reports the frequencies of all PDs for O-BD, O-MDD and O-WELL.

3.3. Differential risk for PD diagnoses in offspring based on maternal Axis I diagnosis

Because of the low frequency of diagnoses present in the sample, modeling the risk for individual PD diagnosis outcomes at each time point was not possible for several of the PDs. Therefore, for the categorical outcome analyses, we created broader outcome variables which included any of the PDs within DSM-IV PD clusters A, B and C for each time point. At T4, O-BD showed a higher prevalence of Cluster B PDs than O-MDD (OR=3.1, p=0.03) and O-WELL (OR=2.4, p=0.08). After correcting for additional potential explanatory variables (SES, maternal GAF, maternal PDs, and offspring mood disorder at T4), O-BD still showed a greater prevalence of cluster B disorders than O-MDD (OR=1.3, p=0.03). Other significant predictors in that model included SES (OR=−0.05, p=0.009) and offspring mood disorder at T4 (OR=1.5, p=0.003). At T5, O-BD showed a higher prevalence of Cluster B PDs than O-WELL (OR=3.7, p=0.04). After correcting for additional potential explanatory variables (SES, maternal GAF, maternal PDs, and offspring mood disorder at T5), this finding was no longer significant. Significant explanatory variables in this model included average family stress (OR=0.44, p=0.03) and offspring T5 mood disorder (OR=3.7, $p < 0.0001$).

3.4. Differential PD symptoms in offspring based on maternal Axis I diagnosis

We evaluated whether dimensional PD outcomes (PD symptom scores) at each time point (T4 and T5) differed by maternal lifetime diagnosis group. In general, most of these analyses revealed the following pattern: O-BD demonstrated the highest (most pathological) scores on PD scales, followed by O-MDD, followed by O-WELL.

Table 2
Frequency of personality disorders in mothers and offspring at T4 and T5.

Frequency of diagnosis present (maternal number: prevalence at T3. Offspring number: first row, maternal prevalence at T3[a], prevalence at T4, as defined by the SNAP-Y and second row, prevalence at T5, as defined by the SCID-II)[b]

PD	Well mothers	Well offspring	Bipolar mothers	Bipolar offspring	MDD mothers	MDD offspring
N attending the visit[c]	26	44 41	23	37 35	39	65 60
Cluster A						
Schizoid	0 (0%)	1 (2%) 0 (0%)	1 (4%)	0 (0%) 1 (3%)	1 (3%)	2 (3%) 0 (0%)
Schizotypal	0 (0%)	0 (0%) 1 (2%)	4 (17%)	0 (0%) 1 (3%)	1 (3%)	1 (2%) 0 (0%)
Paranoid	0 (0%)	1 (2%) 0 (0%)	2 (9%)	2 (5%) 0 (0%)	3 (8%)	2 (3%) 1 (2%)
Cluster B						
Antisocial	0 (0%)	0 (0%) 1 (2%)	1 (4%)	2 (5%) 2 (6%)	3 (8%)	3 (5%) 2 (3%)
Borderline	0 (0%)	3 (7%) 2 (5%)	9 (39%)	5 (14%) 3 (9%)	8 (21%)	4 (6%) 3 (5%)
Narcissistic	0 (0%)	0 (0%) 0 (0%)[d]	1 (4%)	2 (5%) 0 (0%)	2 (5%)	4 (6%) 1 (2%)[b]
Histrionic	2 (8%)	7 (16%) 0 (0%)	5 (22%)	11 (30%) 1 (3%)	5 (13%)	7 (11%) 2 (3%)
Cluster C						
Avoidant	0 (0%)	4 (9%) 1 (3%)	5 (6%)	2 (5%) 0 (0%)	5 (13%)	2 (3%) 1 (2%)
Dependent	1 (4%)	0 (0%) 0 (0%)	3 (13%)	2 (5%) 1 (3%)	3 (8%)	4 (6%) 2 (3%)
Obsessive-compulsive	2 (8%)	0 (0%) 0 (0%)	2 (9%)	0 (0%) 2 (6%)	4 (10%)	2 (3%) 3 (5%)
Multiple PDs	0 (0%)	3 (6.8) 3 5.0)	22 (46%)	5 (13.5) 6 (17.1)	20 (24%)	5 (13.5) 7 (11.9)

SNAP-Y=Schedule for Nonadaptive and Adaptive Personality-Youth Version; SCID-II=Structured Clinical Interview for DSM-IV Axis II Disorders; MDD=Major depressive disorder; and PD=Personality disorder.

[a] PD data from 3 of 91 mothers was missing.
[b] Cases are included in the table if they met full criteria for the disorder (not subthreshold). Percents are computed among those who attended the stated visit.
[c] Numbers for mothers are from T3; numbers for offspring from T4 are in first row, and from t% in second row.
[d] Two of the attending offspring (one O-WELL, one O-MDD) did not provide sufficient data for determination.

Table 3
PD symptoms during mid-adolescence (T4) in offspring groups.

PD	Least squares means and standard errors of *T* scores for symptom scales			Estimated group differences with odds ratios	
	O-WELL	O-BD	O-MDD		
Cluster A					
Schizoid	48.5 (1.2)	51.4 (1.7)	50.5 (1.5)	O-BD vs. O-MDD	0.9
				O-BD vs. O-WELL	2.9
				O-MDD vs. O-WELL	1.9
Schizotypal	45.4 (2.0)	51.7 (1.4)	45.7 (1.5)	O-BD vs. O-MDD	6.0**
				O-BD vs. O-WELL	6.3**
				O-MDD vs. O-WELL	0.3
Paranoid	46.2 (1.9)	53.3 (2.1)	45.3 (1.4)	O-BD vs. O-MDD	8.0**
				O-BD vs. O-WELL	7.1**
				O-MDD vs. O-WELL	−0.9
Cluster B					
Antisocial	46.0 (1.3)	53.2 (2.1)	50.0 (1.3)	O-BD vs. O-MDD	3.2
				O-BD vs. O-WELL	7.2**
				O-MDD vs. O-WELL	4.0*
Borderline	47.4 (1.8)	52.7 (2.4)	47.8 (1.3)	O-BD vs. O-MDD	4.8
				O-BD vs. O-WELL	5.3
				O-MDD vs. O-WELL	0.5
Narcissistic	44.9 (1.4)	49.2 (2.4)	45.4 (1.3)	O-BD vs. O-MDD	3.8
				O-BD vs. O-WELL	4.3
				O-MDD vs. O-WELL	0.5
Histrionic	47.2 (1.7)	48.8 (1.9)	46.0 (1.4)	O-BD vs. O-MDD	2.8
				O-BD vs. O-WELL	1.6
				O-MDD vs. O-WELL	−1.1
Cluster C					
Avoidant	47.1 (1.8)	50.2 (1.6)	47.6 (1.3)	O-BD vs. O-MDD	2.7
				O-BD vs. O-WELL	3.1
				O-MDD vs. O-WELL	0.4
Dependent	46.2 (1.6)	52.0 (2.0)	47.4 (1.4)	O-BD vs. O-MDD	4.6*
				O-BD vs. O-WELL	5.8**
				O-MDD vs. O-WELL	1.2
Obsessive-compulsive	44.2 (1.4)	46.5 (1.4)	44.1 (1.1)	O-BD vs. O-MDD	2.4
				O-BD vs. O-WELL	2.3
				O-MDD vs. O-WELL	−0.1

BD = Bipolar disorder; MDD = Major depressive disorder; and PD = Personality disorder.
Highlighted rows indicate which analyses remained significant after correcting for socioeconomic status, global assessment of functioning, maternal substance use disorder, maternal personality disorder, and offspring mood disorder.

* $p <= 0.05$.
** $p <= 0.01$.

PD symptoms in adolescent offspring (T4) are summarized in Table 3. O-BD exhibited significantly more schizotypal and paranoid PD symptoms than O-WELL and O-MDD. O-BD showed significantly more dependent PD symptoms than O-WELL. Both O-BD and O-MDD showed significantly more antisocial PD symptoms than O-WELL. After correcting for additional potential explanatory variables (SES, maternal GAF, maternal PDs, and offspring mood disorder at T4), the findings with respect to paranoid PD symptoms remained significant, and SES and offspring mood disorder were also significant explanatory variables in the models. Findings for schizotypal and antisocial PD symptoms did not withstand corrections, and again SES and offspring mood disorder were significant explanatory variables. Findings for T4 dependent PD were no longer significant in the more complex model, but none of the additional covariates were significant predictors either.

PD symptoms for young adult offspring (T5) are summarized in Table 4. Both O-BD and O-MDD had significantly more antisocial and obsessive-compulsive PD symptoms than O-WELL. Additionally, O-BD showed more borderline and histrionic symptoms than O-WELL. After correcting for additional potential explanatory variables (SES, maternal GAF, maternal PDs, and offspring mood disorder at T4), findings with respect to antisocial, borderline, and histrionic PD symptoms were no longer significant; offspring mood disorder was the only significant explanatory variable in the models. Findings with respect to obsessive-compulsive PD symptoms were also not significant in the corrected model, and in addition to offspring mood disorders, maternal substance use disorder was also a significant predictor of obsessive-compulsive PD symptoms.

3.5. Continuity of PD symptoms between T4 and T5

Correlations of PD symptom levels between the assessments at T4 and T5 are shown in Table 5. Significant correlations were observed for paranoid, schizotypal, antisocial, borderline, histrionic, avoidant and dependent PD symptoms.

4. Discussion

Longitudinal study of high-risk offspring provides the opportunity to more fully understand the emergence of psychopathology during adolescence and early adulthood. This longitudinal analysis of PDs in offspring of mothers with mood disorders revealed a greater degree of psychopathology in the offspring of mothers with mood disorders compared to low-risk offspring in adolescence and in early adulthood, with O-BD showing the greatest degree of psychopathology.

Table 4
PD symptoms during early adulthood (T5) in offspring groups.

PD	Least squares means and standard errors			Estimated group differences with odds ratios	
	O-WELL	O-BD	O-MDD		
Cluster A					
Schizoid	0.1 (0.1)	0.4 (0.2)	0.2 (0.1)	O-BD vs. O-MDD	0.1
				O-BD vs. O-WELL	0.2
				O-MDD vs. O-WELL	0.1
Schizotypal	0.7 (0.3)	0.6 (0.3)	0.4 (0.3)	O-BD vs. O-MDD	0.2
				O-BD vs. O-WELL	−0.1
				O-MDD vs. O-WELL	−0.3
Paranoid	0.9 (0.3)	0.9 (0.3)	1.0 (0.3)	O-BD vs. O-MDD	−0.1
				O-BD vs. O-WELL	0.1
				O-MDD vs. O-WELL	0.1
Cluster B					
Antisocial	1.5 (0.5)	4.1 (0.8)	3.3 (0.7)	O-BD vs. O-MDD	0.8
				O-BD vs. O-WELL	2.7***
				O-MDD vs. O-WELL	1.8*
Borderline	1.4 (0.4)	2.9 (0.7)	2.3 (0.5)	O-BD vs. O-MDD	0.7
				O-BD vs. O-WELL	1.6*
				O-MDD vs. O-WELL	0.9
Narcissistic	1.0 (0.4)	1.3 (0.4)	1.0 (0.3)	O-BD vs. O-MDD	0.3
				O-BD vs. O-WELL	0.3
				O-MDD vs. O-WELL	0.0
Histrionic	0.9 (0.2)	2.3 (0.6)	1.5 (0.4)	O-BD vs. O-MDD	0.8
				O-BD vs. O-WELL	1.5**
				O-MDD vs. O-WELL	0.7
Cluster C					
Avoidant	1.0 (0.3)	1.2 (0.3)	0.9 (0.2)	O-BD vs. O-MDD	0.2
				O-BD vs. O-WELL	0.1
				O-MDD vs. O-WELL	−0.1
Dependent	0.7 (0.3)	0.9 (0.3)	1.0 (0.3)	O-BD vs. O-MDD	−0.04
				O-BD vs. O-WELL	0.2
				O-MDD vs. O-WELL	0.3
Obsessive-compulsive	1.0 (0.3)	2.6 (0.5)	2.2 (0.4)	O-BD vs. O-MDD	0.4
				O-BD vs. O-WELL	1.6**
				O-MDD vs. O-WELL	1.2*

BD=Bipolar disorder; MDD=Major depressive disorder; and PD=Personality disorder.

* $p <= 0.05$.
** $p <= 0.01$.
*** $p <= 0.001$.

When PDs were assessed categorically, we found greater risk for cluster B disorders in O-BD in adolescence and in early adulthood. When symptoms were viewed dimensionally, we found elevated levels of sub-threshold PD symptoms in several PDs across clusters in offspring of mothers with mood disorders, with O-BD showing the highest levels and O-MDD showing intermediary levels in comparison to controls. This pattern was generally true both in adolescence and in young adulthood, irrespective of whether self-report (T4) or clinical interviews (T5) were used to assess PDs. At T4, several of the findings (cluster B diagnosis, paranoid and schizotypal PD symptoms) withstood correction for numerous additional explanatory variables (SES, maternal GAF, maternal substance use disorder, maternal PD, family stress, and offspring mood disorder). However, none of the T5 findings withstood correction for these additional variables, perhaps due to limited power. Notably, many of the follow-up analyses revealed that the presence of a co-morbid mood disorder (a depressive disorder in adolescence; depression or bipolar disorder in early adulthood) was generally a strong predictor of PD psychopathology, echoing previous work that has underscored an overlap between mood disorder and PD psychopathology [e.g. (Akiskal et al., 1985b; Brieger et al., 2003; Rothen et al., 2009; Weller et al., 1994)].

In this study, O-MDD differed from controls with respect to elevations in antisocial symptoms at T4 and T5, and obsessive-compulsive PD symptoms at T5. These findings add to prior research indicating that O-MDD are at elevated risk for a range of problems. For example, a 2-year longitudinal study reported that O-MDD showed significantly poorer behavioral functioning, social competence, internalizing and externalizing behaviors, and school performance than O-BD or children of medically ill women (Anderson and Hammen, 1993). Similarly, this adds to prior reports from the present work demonstrating significant derailment of development in O-MDD from early childhood through adolescence (Klimes-Dougan et al., 1999; Radke-Yarrow et al., 1992).

The transmission of increased risk for PDs from mothers with mood disorders to their offspring likely reflect an interaction between genetic and environmental influences. Offspring of parents with mood disorders likely inherit shared genes related to both mood disorders and PDs. Core personality dimensions relevant to emotion dysregulation are heritable (Bouchard and Loehlin, 2001). The link identified in the present study between maternal bipolar disorder diagnosis and offspring cluster B personality psychopathology adds to converging evidence suggesting that risks for mood disorders and PDs coincide. Longitudinal studies of patients with borderline PD found elevated incidence of BD (Akiskal et al., 1985a) and of MDD (Links et al., 1995). Studies that have examined family histories of individuals with PDs have found that relatives of those with PDs have higher rates of mood disorders than relatives of individuals without PDs (Akiskal et al., 1985a). Conversely, family histories of those with mood disorders

Table 5
Spearman partial correlations[a] between T4 and T5[b] offspring PD symptom levels.

PD	Correlation
Cluster A	
Schizoid	0.03
Schizotypal	0.19*
Paranoid	0.34***
Cluster B	
Antisocial	0.30**
Borderline	0.41***
Histrionic	0.31***
Narcissistic	0.1
Cluster C	
Avoidant	0.27**
Dependent	0.47***
Obsessive-Compulsive	0.06

PD=Personality disorder.
P-values are two-sided and from a Fisher test of the Spearman correlation at a significance of different *p*-values.

[a] Partial Spearman correlations included correction for SES.

[b] Two of the attending offspring (one O-WELL, one O-MDD) did not provide sufficient data for determination.

* $p <= 0.05$.

** $p <= 0.01$.

*** $p <= 0.001$.

have higher rates of PDs than those without mood disorders (Weller et al., 1994). Furthermore, as noted above our study identified a strong link between the presence of a current mood disorder in the offspring and PD psychopathology.

The environment of the offspring of parents with mood disorders in this study was impacted not only by the presence of a maternal mood disorder and other maternal Axis I and II pathology (e.g., substance abuse disorder, PD[s]), but also (in two-thirds of the cases) by the presence of a paternal psychiatric disorder and by elevated family stress. We found that when correcting for these factors, many of our findings with respect to maternal mood disorder diagnosis were tempered. However, we found little evidence that these factors were significant independent predictors of offspring PD pathology. This may be due to limited power in our study, which was not designed to look at these individual factors as main effects. Further, it is likely that there are other, unmeasured factors associated with living with a mother with a mood disorder that in combination serve to increase risk for PD pathology.

When examining the continuity of offspring PD symptom levels over time, we found significant positive correlations between T4 and T5 assessments for most PDs. However, the strength of these correlations was likely diminished by the difference in methodology used (discussed further below), the length of time between assessments, and the importance of the developmental window in the intervening years. These findings add to a previous longitudinal study of female twins which reported that borderline traits were stable during mid-adolescence and then significantly decreased until the age of 24 (Bornovalova et al., 2009). Similarly, a 2-year longitudinal study of PDs in 15–18-year-olds found low stability of individual PD diagnoses (Chanen et al., 2004). However, when they broadened criteria to examine any PD, they found a high continuity of dimensional PD symptoms (Chanen et al., 2004). Although initial results from the Children in Community study showed persistence of adolescent-diagnosed PDs into adulthood (Kasen et al., 1999), the 20-year outcome results showed that adolescent-onset PDs dissipated over the longer term (Crawford et al., 2008). However, they also found that adolescent PDs predicted poorer functioning on dimensional measures of adult attainment (Crawford et al., 2008). Taken together, this set of findings underscore the value of aggregating PDs and of using dimensional approaches for monitoring personality pathology across time.

4.1. Limitations and future directions

There are several limitations to this study. First, we used different (but developmentally appropriate) instruments to measure PD diagnoses and symptoms: the self-report SNAP-Y at T4 and the clinician-administered SCID-II at T5. Optimally, the same measure might be used to assess a wider developmental window, thus minimizing measurement-dependent biases (e.g. using the adult version of the SNAP in the adult PD assessment battery). Since self-report measures may be more likely to reveal pathology than clinician-administered measures, we may have been more likely to identify pathology during adolescence. For example, we identified a greater prevalence of histrionic PDs across groups at the adolescent visits using the self-report measure than has been typically seen in other population studies. Second, although the sample is large compared to other mood disorder offspring studies, the numbers are still relatively modest. Therefore considering the modest power (especially using categorical measures), group differences that were identified are likely to represent robust group differences. Further, the numbers of participants who completed both T4 and T5 assessments was even smaller, limiting our ability to look at correlations in PD symptomatology across assessments. Third, given that we examined 10 PDs, a conservative approach would be to subject all findings to a Bonferroni correction. In this exploratory study our intent was to minimize Type II error. Future efforts should utilize a larger sample that is more representative of national demographics to increase external validity of the results. Fourth, efforts to account for the variance in the course and timing of each mother's illness may be important to consider with respect to her child's developmental stage. For example, for either mood disorder, severe illness for the mother that occurs in the first year of the offspring's life may be more (or less) disruptive than severe illness that occurs during latency (Toth et al., 2009), including the shaping of temperament and the development of personality pathology. Similarly, chronic illness may be more (or less) disruptive for the child than episodic illness. Future longitudinal work using a fine-grained developmental approach to assess these relationships and to examine the stability of personality traits before, during, and after the emergence of mood episodes, will help clarify the developmental trajectories of comorbid personality and mood disorders.

5. Conclusion

The findings reported here indicate that adolescents and young adult offspring of mothers with mood disorders are at increased risk for PD pathology. The results also suggest that there are some similarities as well as some differences associated with bipolar and unipolar depression. Continued work to characterize the developmental progression of personality psychopathology will be helpful to guide the development of early interventions for these at-risk youths.

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