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Family Functioning as Perceived by Parents and Young Offspring at High and Low Risk for Depression

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Abstract

Background—Family dysfunction has been proposed as one of the environmental mechanisms whereby risk of depression is transmitted from mothers to their children. Using our sample of offspring at high and low familial risk for depression, we hypothesized that: a) high-risk offspring (n = 79) and their mothers will report more extensive family dysfunction than low-risk offspring (n = 82) and their mothers, b) family dysfunction will predict the extent of offspring's depressive symptoms, and c) family dysfunction will mediate the impact of mother's depression on offspring's depressive symptoms.

Methods—The study enrolled 161 offspring of parents who, in a previous study, were ascertained to have either childhood onset mood disorder or no history of a major psychiatric disorder. Parents completed questionnaires and a clinical interview about themselves, their offspring, and the family, while offspring also completed questionnaires about themselves and the family.

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Compliance with Ethical Standards

Ethical approval: All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional research committee and have been performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

Informed consent: Informed consent was obtained from all individual participants included in the study.

Conflict of Interest

There are no conflicts of interest.

Contributors

SD contributed to the coordination of the study, planned the analyses, participated in the interpretation of the data, and drafted the manuscript.

VV contributed to the drafting of the manuscript and interpretation of the data.

KL participated in data collection, identified the topic, and contributed to the drafting of the manuscript;

CG performed the statistical analysis and assisted in data interpretation.

MK conceived the overall study, was responsible for all its aspects, participated in the interpretation of the data, and helped draft the manuscript;

All authors read and approved the final manuscript;

Results—We found support for all three hypotheses. The significant indirect effect between maternal depression and offspring depressive symptoms was driven primarily by offspring's, but not mothers', reports of family dysfunction.

Limitations—Although our assessment of mother's early history of depression was done in a previous study, it is important to note that our results do not inform about causality because of the present study's cross-sectional nature.

Conclusions—The results highlight the importance of detecting and treating family dysfunction, particularly via offspring report, as one way to lower the risk of depression transmission from mothers to their children.

Keywords

maternal depression; family functioning; offspring; high-risk for depression; transmission mechanisms

Given compelling evidence that depression is familial (Beardslee et al., 1998; Birmaher et al., 1996; Neuman et al., 1997; Weissman et al., 1987), a large body of literature has addressed the mechanisms involved in its transmission, particularly from mothers to their juvenile offspring (Goodman & Gotlib, 2002). Children whose parents had histories of depression are at high familial risk for depression throughout their lives, compared to peers whose parents have been free of affective and related psychopathology (see Merikangas & Avenevoli, 2002, for a review). Indeed, parental depression is associated with increased rates of psychopathology among the offspring, particularly depression, and especially so when the parents themselves had juvenile-onset affective disorders (for a review see Beardslee et al., 1998; Goodman & Gotlib, 1999; Grigoroiu-Serbanescu et al., 1991; Hammen et al., 1990; Hops et al., 1990; Lieb et al., 2002; Moldin et al., 1991; Weissman et al., 1988; Weissman et al., 2005). Notably, a meta-analysis revealed that it is maternal depression that has particularly adverse effects (Connell & Goodman, 2002).

In a seminal review paper, Goodman and Gotlib (1999) identified several broad classes of mechanisms that transmit depression risk from mother to child, including family environmental mechanisms. The family plays a clear role in providing the developmental context of childhood, and family-based interventions show promise for improving emotional disorders among children and adolescents (Carr, 2009). Therefore, it is important to understand the potential mechanistic role of family functioning through which parental psychopathology may have negative consequences for offspring. *Family functioning* is a multi-faceted construct that originated from a family system perspective, which views the family as a complex integrated system designed to satisfy the basic needs of its members (Ryan & Keitner, 2009). Although family functioning includes narrower constructs related to parenting behaviors, its emphasis is on the collective health of the family unit (see Miller, Ryan, Keitner, Bishop, & Epstein, 2000; Knafl, Leeman, Havill, Crandell, & Sandelowski, 2015). Following Goodman and Gotlib's (1999) prediction of environmental transmission mechanisms, we tested whether a global index of family functioning mediates the relationship between maternal depression history and child depressive symptoms.

Existing findings support associations between various combinations of our targeted variables. First, maternal depression has been associated with family dysfunction. Both current/recent and past maternal depression compromise the mother's emotional, social, and interpersonal functioning (Angold, 1988; Downey & Coyne, 1990; Rutter, 1989). Mother-child interactions in high-risk families are characterized by more maternal disengagement, lower control, and lower positive affect toward offspring, compared to healthy control families (Dietz et al., 2008; McMakin et al., 2011). Additionally, offspring of depressed parents are exposed to higher rates of marital dysfunction, affectionless control, or low cohesion, which prognosticate poor family functioning (Birmaher et al., 2004; Fendrich et al., 1990; Nomura et al., 2002; Pilowsky et al., 2006; Stein et al., 2000).

Second, there is evidence that negative family environment is related to offspring depressive symptoms (Freed et al., 2016). For example, less supportive and more conflict-ridden family environments are associated with current and future depressive symptoms in children (Sheeber et al., 1997). Offspring experiencing less attachment security and parental approval tend to be more depressed (see Sheeber et al., 2001 for a review). Furthermore, outcomes of family-based interventions for youth depression suggest that family dysfunction and depression in offspring are related to one another (Kaslow et al., 2012; Restifo & Bögels, 2009).

Several studies have set out to test the mediating role of factors related to family dysfunction in the intergenerational transmission of psychopathology. However, most studies were not in a position to assess perceived family dysfunction as a mediator of intergenerational transmission of risk for depression per se, either because they did not predict depressive symptoms of the offspring in particular, or because they examined as mediators constructs other than the perception of family functioning. For example, Johnson and colleagues (2001) followed a nationally representative, middle-class sample, and found that maladaptive parenting behaviors mediated the longitudinal relationship between parental and offspring psychiatric disorders of any kind (see also Elgar et al, 2007 for similar findings). Two cross-sectional studies (Bifulco et al., 2002; Burt et al., 2005) examined samples at elevated risk for psychopathology due to poverty: Burt and colleagues (2005) found a measure of family conflict to mediate the link between mother's lifetime depression severity and children's behavioral problems; Bifulco and colleagues (2002) found that child abuse and neglect mediated the link between maternal depression history and the risk of any major psychiatric disorder. In non-clinical samples, family conflict, parenting and negative emotional expression among family members have been shown to cross-sectionally mediate the link between maternal depressive symptoms and offspring negative affect and adjustment problems (Aunola et al., 2015; Schudlich & Cummings, 2007; Yeh et al., 2016). By contrast, a follow-up study of mothers treated for depression found that the link between maternal remission from depression and offspring internalizing and externalizing symptoms three months later was not mediated by family functioning (Foster et al., 2008).

Only one study has examined family dysfunction as a mediator of the transmission of risk for depression (Garber & Cole, 2010). In a growth model analysis, family dysfunction mediated the relation between maternal depression history and increase in offspring's depressive symptoms over the course of adolescence. However, since maternal depression

and offspring were evaluated, at the same time, it is especially difficult to draw inferences about the direction of relationships. Family dysfunction could have been present prior to the onset of mothers' depression and have contributed to both mothers' and children's depression, rather than serving as a mechanism of transmission.

Several other issues have made it difficult to understand the mediating role of family environment in depression transmission. First, previous studies did not take into account the effect of single-parent households, which have been linked to greater difficulties in family functioning (Amato, 1987; Hayden et al., 1998; McKeown et al., 1997), and thus could influence the transmission of depression. Second, studies concerning depression must consider informants current depression symptoms, which can color their responses. Lastly, the use of single informants may have resulted in an attenuated view of family functioning (Burt et al., 2005; De Los Reyes & Kazdin, 2005). Indeed, perspectives of different family members vary, with offspring tending to adopt more negative perspectives on family functioning than their parents (Noller & Callan, 1986; Tamplin & Goodyer, 2001; Tein et al., 1994).

Building on the findings of previous research, we tested three hypotheses: (1) High-risk families (i.e., with mothers diagnosed with depression) will report more dysfunction compared to low-risk families; (2) Extent of family dysfunction will be related to depressive symptoms of offspring; and (3) Familial transmission of depression to offspring will be partially mediated by the perception of family functioning of both mother and offspring. To strengthen testing of these hypotheses, all our analyses controlled for age, sex, and single-parent household status. We also accounted for current maternal depression symptoms to control for reporting bias. To further minimize informant bias, we used clinicians' ratings to quantify current depressive symptoms of offspring.

Methods

Subjects

The available sample included 246 offspring of parents who, in a previous study, were ascertained to have had either childhood onset mood disorder or no history of any major psychiatric disorder. Parents were recruited between the years 1996 and 2004 for a longitudinal Program Project examining risk factors for childhood-onset mood disorders (Forbes et al., 2005; Miller et al., 2002). For the current analysis, we focused only on identified 161 offspring (85 females) who were younger than 18 years ($M = 11.99$, $SD = 2.83$), had normal IQ, and whose mothers were the probands ($n = 46$; meeting DSM criteria for major depression or dysthymia by age 14.99 and no subsequent bipolar disorder) or controls ($n = 43$; free of any major psychiatric disorder with both juvenile and adult-onset). Proband mothers had 79 children, henceforth called "high-risk offspring" and control mothers had 82 children, henceforth called "low-risk offspring". Of the 89 families in the study, 38 participated with one child, 32 with sibling pairs, 17 with three siblings, and 2 with four siblings. Not all siblings in a family participated on the same day. Ninety-four percent of mothers had a high school diploma and 30% were unemployed at the time of their interview. The racial make-up of offspring was 66% Caucasian, 22% African American, and 11% Biracial. Fifty-three percent were from intact families.

At the time of their interview for the present study, 21% of mothers (41% of probands and zero controls) were in a depressive episode, and 24% (46% of probands and zero controls) had a current anxiety disorder. Thirty three percent of proband mothers currently had both a depressive and an anxiety disorder. In turn, 7% of the offspring (11% of high-risk and 2% of low-risk) were in a depressive episode, 11% (19% of high-risk and 2% of low-risk) had a current anxiety disorder, and 4% (8% of high risk and 1% of low risk) were in both anxiety and depressive episodes 13% (20% of high-risk and 6% of low-risk) had a current diagnosis of attention-deficit/hyperactivity disorder and 7% (10% of high-risk and 4% of low-risk) had current oppositional defiant disorder or conduct disorder.

Recruitment and Procedure

All mothers had participated in a previous Program Project during which the onset of their depression was established as prior to age 14.99. Diagnoses were confirmed via the Semi-Structured Clinical Interview for DSM-IV (SCID; First et al., 1995) by trained professional clinicians. As described in detail elsewhere (Forbes et al., 2006), diagnoses were reviewed by pairs of psychiatrists who gave ‘best estimate’ consensus diagnoses (Maziade et al., 1992) based on all psychiatric and medical records. In the current study, mothers again were interviewed via the SCID (First et al., 1995). Furthermore, parents and offspring were interviewed separately about the child’s psychiatric status via the Interview Schedule for Children and Adolescents-Diagnostic Version (ISCA-D), a DSM-IV based variation of the original schedule (Sherrill and Kovacs, 2000). Parents completed questionnaires and a clinical interview about themselves, their offspring, and the family, while offspring also completed various self-rated scales about themselves and the family. Informed parental consent and offspring assent were obtained prior to study participation.

Measures

Maternal depression history—The Semi-Structured Clinical Interview for DSM–IV Axis I Disorders, Patient Edition (SCID; First et al., 1995) was used to assess lifetime psychiatric disorders among mothers. The SCID was expanded to include criteria for selected childhood diagnoses. In the previous study, we found the interrater reliability of mood disorder diagnoses to be good to excellent (*ICC*: .66 – .91).

Mothers also completed the Beck Depression Inventory-II (BDI-II; Beck et al., 1996), a 21-item inventory to assess the severity of current depressive symptoms. The BDI-II had excellent internal reliability in the present sample (α = .93).

Offspring’s symptoms—Clinicians’ ratings of offspring’s depressive symptoms were obtained via the ISCA-D, the semi-structured diagnostic interview. The clinician rated each of 17 current/recent symptoms on a scale from 0 to 2, and these ratings were summed to create a continuous depression severity score from 0 to 34. Interviewers were trained clinicians with a Master’s or Doctoral degree. The ISCA-D has previously shown acceptable interrater reliability in children and adolescents, ranging from .63 to .92 for current depression from child interviews and .65 to .87 from parent interviews (Kiss et al., 2007).

Family functioning—To assess family functioning, mothers and their children independently completed the 12-item General Functioning scale of the McMaster Family Assessment Device (FAD; Epstein et al., 1983). The general functioning scale quantifies the overall emotional health and functioning of the family through questions such as “We confide in each other” and “There are lots of bad feelings in our family” (Epstein et al., 1983). Respondents record their answers on a 4 point, Likert-type scale, from 1 = Strongly Agree to 4 = Strongly Disagree. Epstein and colleagues (1983) reported the FAD general functioning scale to have high reliability as well as good concurrent and predictive validity (see also Byles et al., 1988). The FAD also discriminates between clinical and non-clinical samples (Kabacoff et al., 1990). In nonclinical samples, the general functioning score mean was 1.96 while clinical samples had a mean score of 2.26 with a pooled standard deviation of 0.53 (Epstein et al. 1983).

Statistical Procedures

Discrete and continuous variables were compared in high and low-risk groups using χ^2 and *t*-tests for independent samples. Bivariate relationships were examined using Pearson correlations. To quantify the relationship between symptom severity and family functioning, multiple regression analysis via a mixed-effects model was employed to control for risk-status, age, sex, single-parent household, and the random effect of family clusters (see below). Multiple regression analyses were also performed to test the mediating role of maternal and offspring’s perceptions of family functioning in the relationship between familial risk and depressive symptoms in offspring. Specifically, the bootstrapping technique for testing multiple mediation was used (Preacher & Hayes, 2008; VanderWeele & Vansteelandt, 2014). This approach provides indirect effects estimates that can be used to make decisions regarding whether significant mediation is occurring in models with multiple mediators.

To account for sibling relationships in our design, family clustering was corrected in any group comparisons of offspring via Taylor Series linearization (Woodruff, 1971), and in any mixed-effect regression model via the method of Kenward and Roger (Kenward & Roger, 2009) for adjusting variance and degrees of freedom. Effect sizes for these data are reported in terms of φ for Rao-Scott χ^2, *d* for *F* statistics in group comparisons, and partial R^2_{β} for *F* statistics in mixed models.

Results

Descriptive Statistics

Descriptive results appear in Tables 1 and 2. The two groups of offspring did not differ in sex ratio (χ^2 (2, N = 161) = 0.11, $p > .9$, $\varphi = 0.03$). Although the low-risk sample had more African Americans compared to the high-risk group, this difference was not significant after accounting for family clusters (Rao-Scott χ^2 (2, N = 161) = 2.19, $p = .34$, $\varphi = 0.12$). Mothers of low-risk offspring were significantly older than mothers of high-risk offspring ($F(1,88) = 6.76$, $p = .011$, $d = 0.55$). In addition, 1 (1%) low-risk and 8 (10%) high-risk offspring were in single-parent households (Rao-Scott $\chi 2$ (2, N = 161) = 5.44, p = .02, φ = 0.18). Single-parent household status correlated with reports of general family functioning

by offspring (r = .16, p = .049) and mothers (r = .17, p = .003), but not with offspring depressive symptoms (r = .08, p = .29; see Table 2).

High-risk offspring had more depressive symptoms than low-risk offspring ($F(1,88) = 17.50$, $p < .001$, $d = 0.89$; see Table 1). Offspring ratings of family dysfunction within families were independent ($ICC_{CHILD} = .05$), but maternal ratings within family were correlated ($ICC_{MOTHER} = .88$). Offspring's and mothers' reports of family dysfunction were significantly correlated with each other ($r = .26$, $p < .001$; see Table 2), and this association did not differ between high- and low-risk groups ($F(1,101) = 0.19$, $p > .6$, $d = 0.09$).

Maternal Depression Histories and Family Functioning

Confirming our first hypothesis (see Table 1), high-risk and low-risk families differed in their reports of general family functioning. The effect of group status was significant among both offspring ($F(1,88) = 4.28$, $p = .042$, $d = 0.44$) and mothers ($F(1,88) = 11.28$, $p = .045$, $d = 0.72$). The direction of effects was such that offspring at high risk for depression and their mothers perceived their families as more impaired than did low-risk offspring and their mothers.

Paired sample *t*-tests revealed a significant difference between offspring's and mothers' reports of family function ($t(88) = 2.72$, $p = .008$, $d = 0.58$), suggesting that offspring see their families as more dysfunctional than their mothers do ($M_{CHILD} = 1.92$, $SD_{CHILD} = 0.57$, $M_{MOTHER} = 1.76$, $SD_{MOTHER} = 0.49$). The parent-child difference did not vary by risk status ($F(88) = 1.67$, $p = .20$, $d = 0.28$).

Family Functioning and Offspring's Depressive Symptoms

Without statistical adjustment, both informants' (offspring and mothers) reports of family dysfunction were positively correlated with offspring's depressive symptoms ($r_{CHILD} = .33$, $p < .001$; $r_{MOTHER} = .38$, $p < .001$; see Table 2). To examine the second hypothesis, a regression analysis was conducted with both informants' reports of family dysfunction as predictors of offspring's clinically rated depressive symptoms, controlling for family effect, age, sex, and single-parent household. This analysis revealed that both offspring's and mother's reports of family dysfunction independently and additively accounted for variance in offspring depression (offspring: $\beta = 1.79$, SE = 0.46, $p < .001$, partial $R^2_{\beta} = .13$; mothers: $\beta = 2.16$, SE = 0.78, $p = .007$, partial $R^2_{\beta} = .08$).

We then considered maternal parameters that could explain these reports in two different models. Upon adding risk status to the regression model, high risk significantly predicted elevated symptoms ($\beta = 3.27$, SE = 0.87, $p < .001$, partial $R^2_{\beta} = .17$). Family functioning according to offspring report remained significant ($\beta = 1.69$, SE = 0.46, $p < .001$, partial $R^2_{\beta} = .12$) and maternal report of family functioning dropped to a trend level ($\beta = 1.43$, SE = 0.75, $p = .06$, partial $R^2_{\beta} = .05$). Replacing risk status with current depressive symptoms of the mother (BDI-II scores) in the model, current maternal symptoms significantly predicted elevated symptoms in offspring ($\beta = 0.14$, SE = .04, $p < .001$, partial $R^2_{\beta} = 0.11$). Family dysfunction according to the offspring remained significant ($\beta = 1.68$, SE = .48, $p < .001$, partial $R^2_{\beta} = 0.11$), but mother's report of family functioning dropped to a trend level ($\beta = 1.36$, SE = 0.77, $p = .08$, partial $R^2_{\beta} = .03$).

Indirect Effects of Depression Risk Status on Offspring Depression Severity via Family Functioning

We then tested whether family functioning scores explain the association between offspring's depression risk status and their depressive symptom severity, using a multiple mediation approach (VanderWeele & Vansteelaundt, 2014) with Monte Carlo confidence intervals (CI) (MacKinnon et al., 2004). In this approach, mediators are interpreted as significant if the point estimate of the indirect effect differs significantly from zero, or in other words, if the 95% confidence interval (CI) does not contain zero. We entered both offspring's and mothers' family functioning reports into the statistical model simultaneously as potential mediators. Results revealed that the total indirect effect of both mother and offspring reports of family functioning was significant (0.87; 95% CI = 0.19 to 1.72). Of these, the specific indirect effect of offspring report of family functioning was statistically significant (0.39; 95% CI = 0.07 to 0.83), while the specific indirect effect of maternal report of family functioning was not (0.48; 95% CI = −0.11 to 1.22).

Discussion

There is broad agreement that multiple mechanisms are involved in the intergenerational transmission of depression from mothers to their children. In order to contribute to that literature, we tested three hypotheses focusing on the potential mediating role of family environment. Confirming our first hypothesis, we found that offspring at high familial risk for depression and their mothers perceived their family environments as more impaired than did low-risk offspring and their mothers. Second, the elevated levels of family dysfunction predicted offspring's depressive symptoms above and beyond the predictive value of familial risk status. Finally, family dysfunction reported by the offspring partially mediated the association between offspring's familial risk status and their depressive symptoms. Overall, these findings support the likelihood that family dysfunction serves as one mechanism of the intergenerational transmission of depression.

Our comparison of both mothers' and offspring's reports of family functioning is important, given the restricted perspective associated with a single informant report (Burt et al., 2005). In our sample, mothers' and offspring's family functioning reports correlated significantly with one another. At the same time, the magnitude of this correlation was modest ($r = .26$), suggesting each informant had a distinct perspective on the health of the family unit. Furthermore, we observed that, while the total indirect effect on offspring depression was significant when both indices were considered together, it was driven largely by offspring's reports of family dysfunction. Only offspring's reports of family dysfunction emerged as an independent mediator above and beyond the mediating role of mothers' reports of dysfunction. If our dependent variable of offspring depression severity had been assessed via self-report, we might have suspected that method or informant variance was inflating the mediation. However, our offspring depression outcomes were assessed by trained clinicians, which can bolster confidence in our mediation result. If future studies replicate this unique mediating role of offspring–perceived family dysfunction, then offspring's reports should be taken seriously as markers of risk for intergenerational depression transmission.

Our finding regarding the transmission of familial depression is noteworthy because the onset of maternal depression clearly predated the birth of the offspring and was not a reaction to it. Thus, we established the temporal precedence of maternal depression with respect to depression in her offspring, considerably improving upon and extending the literature. If our results are replicated using a more optimal design, one implication will be that improving family functioning is a viable way to help interrupt the effects of depression in affected families.

Given that family environment itself can be affected by several other factors, in the future it will be important to examine moderating influences. For example, family dysfunction may be more harmful during the pre-school years as compared to adolescence (Goodman & Gotlib, 1999). Due to the narrow age range of children in the present sample, we were not able to examine this question. Other moderating influences may include protective factors in the environment such as social support. Family dysfunction may be more harmful for children with a lack of a social support system, as compared to children who have a stronger social support system (Gauze et al., 1996). Thus, a social support system may buffer against the harmful effects of family dysfunction. Therefore, both mediational and moderational approaches should be taken into account in order to understand the mechanisms of depression transmission.

Our study had several strengths, including the specificity of risk status, the emphasis on multi-informant assessment of family functioning, the use of clinician's ratings of offspring' depressive symptoms, and controlling for single-parent household. However, the results should also be interpreted in the context of some limitations. First, our assessment tool of family functioning was global and did not provide information about which specific aspect of family life is dysfunctional. Furthermore, we only examined perceived and not actual family functioning. Future studies should consider more objective assessment of family functioning among high- and low-risk families, incorporating performance-based family tasks and naturalistic observations. Second, we considered only maternal history of depression as a familial risk factor and only the maternal perspective on family functioning. Future work should consider the impact of fathers as well. Finally, our assessment of mother's early history of depression took place in a previous study, making their assessment independent and separated in time from the assessment of the child's depressive symptoms. However, our results do not inform about causality because of the current study's correlational and essentially cross-sectional nature. Future studies should consider a longitudinal design to better infer temporal relationships (Maxwell & Cole, 2007).

Overall, this study contributes to the growing literature on environmental mechanisms of the intergenerational transmission of depression. Our study highlights the importance of family dysfunction and suggests that efforts to prevent depression onset among vulnerable children and youths, should be invested in improving the family environment.

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Highlights

- We examined family functioning as a mechanism of depression transmission.
- High-risk offspring and their mothers reported more family dysfunction than low-risk offspring and their mothers.
- Family dysfunction predicted the extent of offspring's depressive symptoms.
- Family dysfunction mediated the link between mother's and offspring's depression.

Table 1

Demographic Characteristics and Means (Standard Deviations) for Self-report Measures

	High-Risk (n=79)	Low-Risk (n=82)	Effect size
Age of offspring	11.50 (2.83)	12.46 (2.78)	0.47*
Age of mother	36.44 (4.47)	39.45 (5.87)	0.55**
Sex ratio (F/M)	42/37	43/39	0.03
Race (B/A/C)	10/12/57	8/24/50	0.12*
Single-parent household, n (%)	8 (10)	1 (1)	0.18
FAD offspring	2.02 (0.53)	1.83 (0.60)	0.44*
FAD mother	1.93 (0.47)	1.59 (0.45)	0.72**
Offspring depressive symptoms	5.62 (5.17)	2.41 (3.28)	0.88**
Maternal depressive symptoms	15.11 (11.85)	2.89 (4.35)	1.38**

Note. Race: B = Biracial, A = African American, C = Caucasian; FAD = Family Assessment Device; Offspring depressive symptoms = Clinician-assessed depressive symptoms using ISCA-D; Maternal depressive symptoms = Beck Depression inventory II. Effect sizes for differences between high- and low-risk groups were computed based on the particular statistics used for each variable.

* $p \leq .05$;

** $p \leq .01$

Table 2

Pearson Correlation coefficients among key variables (n = 161).

	variables								
	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**
1. High Risk family status (1 = yes)	1	−.28***	−.17*	.01	.19*	.57***	.35***	.16*	.35***
2. Age of mother		1	.32***	−.07	−.00	−.32***	−.09	−.01	−.01
3. Age of offspring			1	.08	.04	−.15	.17*	.21**	.01
4. Offspring's Sex (1 = Female)				1	.18*	.01	.22**	.17*	.05
5. Single-Parent Household (1=yes)					1	.05	.08	.16*	.17*
6. Maternal depressive symptoms						1	.44***	.08	.44***
7. Offspring depressive symptoms							1	.33***	.38***
8. FAD offspring								1	.26**
9. FAD mother									1

*p<.05,

**p<.01,

***p<.001

Maternal depressive symptoms = Beck Depression inventory II; Offspring depressive symptoms = Clinician-assessed depressive symptoms using ISCA-D; FAD = Family Assessment Device General Functioning Scale.