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Psychosocial Impairment in Offspring of Depressed Parents

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Abstract

Background—Offspring of depressed parents experience impairment in a number of domains of functioning. Few studies have examined the impact of both maternal and paternal depression and comorbid psychopathology on offspring functioning.

Method— Participants in the Oregon Adolescent Depression Project (OADP) were administered diagnostic interviews and completed measures of psychosocial functioning during adolescence and again during young adulthood. Diagnostic interviews were also conducted with the mothers and fathers of the target individual.

Results— After controlling for relevant demographic characteristics, parental comorbid psychopathology, and offspring psychopathology, maternal depression was associated with greater levels of physical symptoms during adolescence, and with higher levels of minor stressors and a greater risk for using mental health services in young adulthood. Paternal depression was associated with offspring experiencing more major stressors, having lower perceived social competence, and being at greater risk for suicide attempts during adolescence, as well as lower perceived social competence in young adulthood.

Conclusions— Offspring of depressed parents demonstrate impairment in a variety of domains, regardless of the presence of their own psychopathology. Further research on the mechanisms that lead to these impairments, as well as research that examines the role of these psychosocial impairments in the subsequent development of psychopathology is warranted.

Offspring of parents with major depressive disorder (MDD) exhibit psychosocial impairment in a variety of domains, including academic, peer, and family functioning (Beardslee et al., 1998; Goodman & Gotlib, 2002; Weissman et al., 1997). In addition to being important in its own right, this impairment may mediate the intergenerational transmission of depression from parents to offspring (Cicchetti & Toth, 1998; Goodman & Gotlib, 1999), and has implications for later economic productivity, and interpersonal and family functioning in adulthood. Moreover, even if the offspring do not themselves develop depression, their impairments may place *their* offspring at risk for depression (Warner et al., 1999).

This paper focuses on impairment in the adolescent and young adult offspring of parents with depressive disorders. There is an extensive literature documenting impairment in multiple areas of functioning in the adolescent offspring of depressed parents. Maternal depression has been associated with offspring delinquency, alcohol problems, academic difficulty, and interpersonal conflict (McCombs-Thomas & Forehand, 1991; Davies & Windle, 1997; Shiner & Marmorstein, 1998; Hammen & Brennan, 2001). Paternal depressive symptoms are associated with lower cognitive competence and greater father-child conflict (McCombs-Thomas & Forehand, 1991; Kane & Garber, 2004).

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Fewer studies have examined the effect of parental depression on young adult psychosocial functioning. Birmaher et al. (2004) found few differences between offspring of depressed parents with no personal history of psychopathology and healthy controls. Ensminger et al. (2003) conducted a 25-year follow up of an inner-city cohort of first-graders. They found that sons, but not daughters, of mothers who reported dysphoric mood while the child was in first-grade and again in adolescence were at increased risk for not completing high school.

The literature on the association between parental depression and offspring impairment is limited in a number of ways. Many studies investigate only one domain of offspring functioning. In addition, many combine children in different developmental periods, which is problematic as different competencies are central at different times. Investigations focus mostly on maternal depression, with only a minority of studies including paternal depression (Connell & Goodman, 2002). Lastly, offspring of parents with other forms of psychopathology (anxiety or substance use disorders [SUD]) are also at risk for impaired functioning. However, few studies of the relationships between parental MDD and offspring impairment have included comparison groups of offspring of parents with other forms of psychopathology.

The Oregon Adolescent Development Project (OADP; Lewinsohn et al., 1993) has collected data on both diagnostic and psychosocial outcomes in a large community sample of high school students and followed them through age 24. During adolescence and young adulthood, the OADP assessed multiple domains of functioning that are important in both adolescence and young adulthood (Roisman et al., 2004). A number of these domains have previously been associated with parental MDD, including academic attainment, social competence, peer support, and family support. Mental health utilization and suicide attempts were also assessed. At the time of the age 24 assessment, the biological parents were interviewed concerning lifetime history of psychopathology.

This paper focuses on the impact of parental psychopathology on offspring psychosocial functioning during adolescence and young adulthood. We address the following issues: (1) do maternal and paternal MDD have unique effects on offspring functioning?; (2) does parental depression have a greater impact on some areas of offspring functioning than others?; (3) is functioning affected similarly in adolescence and young adulthood?; (4) are the effects specific to depression, or are they also associated with other forms of parental psychopathology?; (5) do these effects persist even after controlling for psychopathology in the offspring?; and (6) does offspring gender moderate the association between parental psychopathology and psychosocial functioning?

Only limited data are available to provide specific hypotheses concerning these issues. The few studies that have examined the effects of both maternal and paternal MDD on offspring have generally found stronger associations for maternal MDD (Connell & Goodman, 2002). Thus, we expect to find stronger associations between maternal MDD and offspring functioning compared to paternal MDD. During the transition from adolescence to young adulthood, offspring become more independent, often leave home, and focus more and more on peer and romantic, rather than family, relationships. Thus, the influence of family factors may be weaker in young adulthood than in adolescence. Consistent with this, Connell and Goodman (2002) found that the association between maternal MDD and offspring outcomes was larger in younger offspring. Therefore, we expect to find more associations between parental psychopathology and offspring functioning during adolescence than young adulthood. We did not have specific hypotheses regarding the remaining issues.

Method

Participants

Offspring—Offspring were randomly selected from nine high schools in western Oregon. A total of 1,709 adolescents (ages 14–18; mean age 16.6, $SD = 1.2$) completed the initial (T_1) assessments between 1987 and 1989. The participation rate at T_1 was 61%. Approximately one year later, 1,507 of the adolescents (88%) returned for a second evaluation (T_2). Differences between the sample and the larger population from which it was selected, and between participants and those who declined to participate or dropped out of the study before T_2 , were small (Lewinsohn et al., 1993).

At age 24, all adolescents with a history of MDD by T_2 ($n = 360$) or a history of non-mood disorders ($n = 284$), and a random sample of adolescents with no history of psychopathology by T_2 ($n = 457$) were invited to participate in a third (T_3) evaluation. All non-white T_2 participants were retained in the T_3 sample to maximize ethnic diversity. Of the 1,101 T_2 participants selected for a T_3 interview, 941 (85%) completed the age 24 evaluation. The T_2 diagnostic groups did not differ on the rate of participation at T_3 .

At each evaluation, participants completed a questionnaire assessing multiple domains of functioning that was returned by mail. Given our focus on functioning during early adulthood (near the time of the 24th birthday), participants who had not completed a mailer questionnaire during the 23 to 25 age interval ($n = 24$) were excluded from the analyses.

Parents—We assessed lifetime psychopathology in the biological parents of the OADP participants near the time of the T_3 evaluation. When parents could not be directly interviewed, informant data were obtained from the OADP participants, and, if possible, from a second informant (generally the co-parent or a sibling). Of the 1,101 offspring selected for a T_3 interview, diagnostic information on at least one parent was available for 801 (72.7%). Of the 917 offspring with T_3 questionnaire data, parent diagnostic data were available for 770 (84%).

Offspring diagnoses were based on *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edition revised (DSM-III-R; American Psychiatric Association, 1987) criteria; parent diagnoses were based on *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV; American Psychiatric Association, 1994) criteria. Families with parental history of a non-affective psychotic disorder, bipolar disorder, or dysthymic disorder with no lifetime history of MDD were excluded ($n = 59$). Additionally, as all analyses required both parents, all data presented are for offspring who have data on both parents. The final sample consisted of 711 mother, father, and offspring triads. Direct interviews were obtained from 75.2% of the mothers and 46.6% of the fathers.

Diagnostic Interviews

Offspring—At T_1 , offspring were interviewed with a version of the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS; Orvaschel et al., 1982), which combined features of the Epidemiologic and Present Episode versions, and included additional items to derive DSM-III-R diagnoses. At T_3 , offspring were interviewed using the Longitudinal Interval Follow-up Evaluation (LIFE; Keller et al., 1987), which elicited detailed information about the onset and course of psychiatric disorders since the previous evaluation.

To assess interrater reliability, independent raters reviewed audiotapes of a random sample of interviews (N 's ranged from 166 to 233). The reliability of lifetime diagnoses was fair-excellent for MDD (Kappa [K] for T_1 and T_3 was .86 and .86, respectively), any anxiety disorder (K for T_1 and T_3 was .53 and .84, respectively), and any substance use disorder (K for T_1 and T_3 was .89 and .81, respectively).

Parents—Parents were interviewed using the Structured Clinical Interview for DSM-IV Axis I Disorders, non-patient version (SCID-NP; First, et al., 1996). All interviews were conducted without knowledge of the offspring's diagnoses. Independent raters reviewed audiotapes of 184 randomly selected interviews. The interrater reliability of lifetime diagnoses were excellent for MDD ($K = .94$), any anxiety disorder ($K = .90$), alcohol abuse/dependence ($K = .86$), and drug abuse/dependence ($K = .89$).

Family history data were collected using the Family Informant Schedule and Criteria (FISC; Mannuzza & Fyer, 1990), supplemented with all items necessary to derive DSM-IV diagnoses. Independent raters reviewed audiotapes of 242 randomly selected informant interviews. The interrater reliability of lifetime diagnoses were good-excellent for MDD ($K = .90$), any anxiety disorder ($K = .77$), alcohol abuse/dependence ($K = .90$), and drug abuse/dependence ($K = .82$). Consistent with previous research (Chapman et al., 1994), sensitivity of informants' reports of family history was moderate and specificity was high (Klein et al., in press).

Offspring interviews at T_3 and interviews with relatives were conducted by telephone, which generally yields comparable results to face-to-face interviews (Rohde et al., 1997; Sobin et al., 1993). Most interviewers had advanced degrees in a mental health field and several years of clinical experience. Interviewers were trained in the use of the diagnostic instruments and completed a minimum of two supervised training interviews, achieving $K = .80$ or better for concordance between their symptom ratings and those of the supervisor.

As multiple data sources were available for most parents, we derived lifetime best-estimate DSM-IV diagnoses from all available information (Leckman et al., 1982). Two diagnosticians, from a team of four senior clinicians, independently derived best-estimate diagnoses without knowledge of offspring diagnoses. Disagreements were resolved by consensus. Interrater reliability of the independently derived best-estimate diagnoses prior to the resolution of discrepancies was excellent for MDD ($K = .91$), any anxiety disorder ($K = .94$), alcohol abuse/dependence ($K = .97$), and drug abuse/dependence ($K = .96$).

To ensure that the temporal direction of the associations between parental and offspring outcomes were unambiguous, a disorder was considered present in the parent only if the onset preceded T_1 . If the parental disorder first appeared after the offspring entered the study, it was considered absent. This is a conservative approach that slightly attenuates the associations between parent psychopathology and offspring outcomes. Using this criterion, 205 (28.8%) of the mothers and 117 (16.5%) of the fathers had MDD (with or without comorbid non-affective psychopathology), and 226 (31.8%) of the mothers and 330 (46.4%) of the fathers had non-affective disorders (NAD; either anxiety or substance use disorders; with or without comorbid MDD).

Assessment Instruments

Psychosocial constructs—An extensive battery of psychosocial measures was administered to all participants at T_1 and T_3 (see Lewinsohn et al., 1993; Lewinsohn, Roberts et al., 1994; Lewinsohn et al., 2003).

GPA was assessed via offspring self report and was the grade point average of the most recently completed grading period. This was assessed only at T_1 .

School problems were assessed by asking: (a) how many times per week was homework not completed?; (b) how many times in the past month the child did not attend school? and; (c) has the child repeated a grade? Responses to questions (a) and (b) were standardized. The scale was computed as the mean of these three indicators. This was assessed only at T_1 .

Education level attained assessed the number of years of school completed. This was reported for T₃ only.

Unemployment was assessed as the number of weeks unemployed in past year. This was reported for T₃ only. Offspring who attended school were not considered unemployed.

Income was reported as the annual household income earned in the home of the offspring. This was reported for T₃ only.

Living with a biological parent was included as an outcome during offspring young adulthood. This was assessed from the household composition data from the LIFE.

Social support: Family assessed enjoyable and aversive interactions with family members on the basis of items from the Appraisal of Parents subscale of the Conflict Behavior Questionnaire (11 items; Prinz et al., 1979), the Parent Attitude Research Instrument (6 items; Schaefer, 1965), the Cohesion subscale of the Family Environment Scale (3 items; Moos, 1974), the Arizona Social Support Interview Schedule (Barrera, 1986), and the Social Competence Scale of the Youth Self-Report (2 items; Achenbach & Edelbrock, 1987) for the T₁ assessment ($\alpha = .77$). At T₃, this construct was assessed using the Perceived Social Support Questionnaire (10 items; Procidano & Heller, 1983) ($\alpha = .88$).

Social support: Friends assessed the number of friends, frequency of interaction, and relationship quality on the basis of items from the Social Competence Scales of the Youth Self-Report (3 items; Achenbach & Edelbrock, 1987), the UCLA Loneliness Scale (8 items; Russell et al., 1980), and the number of friends listed as providing social support (Barrera, 1986) for the T₁ assessment ($\alpha = .72$). At T₃, this construct was assessed using the Perceived Social Support Questionnaire (10 items; Procidano & Heller, 1983) ($\alpha = .88$).

Stress: Daily hassles assessed the frequency of unpleasant social and nonsocial events in the past month (20 items; Unpleasant Events Schedule, Lewinsohn et al., 1985) ($\alpha = .79$). The same measure was used at both T₁ and T₃.

Stress: Major life events in the year preceding T₁ assessed the occurrence of 14 negative life events to self or significant others (i.e., parent, sibling, other relative, or close friend; selected from the Schedule of Recent Experiences, Holmes & Rahe, 1967; Life Events Schedule, Sandler & Block, 1979). At T₃, this construct assessed 33 events occurring to the offspring or significant others (i.e., parent, sibling, other relative, or close friend) selected from the Social Readjustment Rating Scale (Holmes & Rahe, 1967), and the Psychiatric Epidemiology Research Inventory (Dohrenwend et al., 1986) in the year preceding T₃. We did not include items that may represent symptoms of psychopathology, or if the event did not have a direct impact on the offspring (e.g. sibling or close friend becoming pregnant).

Self-rated social competence assessed self-perceived social competence (5 items; Social subscale of the Perceived Competence Scale for Children, Harter, 1982; 7 items from Lewinsohn et al., 1980; $\alpha = .81$) at T₁. At T₃, self-rated social competence was assessed using only the latter measure ($\alpha = .79$).

Physical health and illness assessed the number of days spent in bed as a result of illness in the past year (1 item), the number of visits to a physician in the past year (1 item), and the occurrence of 88 physical symptoms (e.g., broken bones, ulcers, double vision) during the past 12 months at T₁ ($\alpha = .51$). At T₃ this construct was measured differently (4 items assessing self-rated health, number of times received treatment in past year, treatment for illness or injury in past year, chronic medical problems distress) ($\alpha = .50$).

Suicide attempts were defined as answering affirmatively to the question, “Have you ever tried to kill yourself or done anything that might have killed you?” For more details, see Lewinsohn, Rohde and Seeley (1994). For the T₁ assessment, this variable indicates whether a suicide attempt occurred at any time during the life of the offspring. For the T₃ assessment, this variable indicates whether a suicide attempt occurred between the T₂ and T₃ assessment.

Mental health utilization (MHU) was assessed during the diagnostic interviews at both assessments. Participant reported whether or not they had used mental health services in the past 12 months.

Life satisfaction was assessed at T₃ only with 15 items chosen from Andrews and Withey (1976) and Campbell et al. (1976) ($\alpha = .89$).

Data analysis

Caucasian OADP participants with no history of psychopathology through T₂ were undersampled in the T₃ follow-up, hence in all statistical analyses offspring were weighted as a function of their probability of being selected at T₃. However, numbers and proportions of participants, which are presented for descriptive purposes, are unweighted. Analyses were conducted using SUDAAN (Shah et al., 1997).

Results

Of the offspring, 426 (60%) were female, 405 (57%) resided with both of their biological parents at T₁, 185 (26.0%) met criteria for MDD, 80 (11.3%) met criteria for an anxiety disorder, 72 (10.1%) met criteria for a substance use disorder (SUD), and 51 (7.2%) met criteria for a disruptive behavior disorder (DBD) at the T₁ assessment. At the T₃ assessment, 369 (51.9%) met criteria for MDD, 155 (21.8%) met criteria for an anxiety disorder, 282 (39.7%) met criteria for SUD, and 71 (10.0%) met criteria for DBD. The rates of psychopathology are all lifetime through that assessment. The prevalence rates are inflated at T₃ due to oversampling offspring with psychopathology at T₁ and T₂.

Regression analyses

We examined three sets of models using multivariate regression analyses. The first set of models examined the independent effects of maternal and paternal MDD on the measures of offspring functioning in adolescence and young adulthood. Maternal and paternal interview status (i.e. whether the parent was directly interviewed), offspring gender, highest education completed by either parent (coded as BA/BS achieved vs. not achieved), and living with both biological parents at T₁ were entered as covariates for all of the outcomes explored except for living with a biological parent at T₃. For this variable, the analysis was limited to those offspring who were living with both of their biological parents at T₁. The major variables of interest, maternal and paternal MDD, were entered simultaneously with the covariates.

The second set of models examined the specificity of the effects of parental MDD on offspring functioning. In these models, maternal and paternal MDD, and maternal and paternal non-affective disorders (NAD) were entered simultaneously along with the same set of covariates. The NAD category included anxiety disorders and SUD.

The third set of models examined whether the effects of parental psychopathology on offspring functioning could be accounted for by psychopathology in the offspring. In these models, offspring psychopathology (offspring lifetime diagnosis of MDD, anxiety disorders, SUD, and DBD up to the time of the relevant assessment) was added to the previous set of independent variables.

T₁ outcomes—Data for these analyses are summarized in Table 1. When controlling for interview status of both mothers and fathers and demographic covariates (offspring sex, living with both biological parents at T₁, and parental education), maternal MDD was not associated with any of the outcomes examined. Paternal MDD was associated with lower GPA, more minor stressors, more major stressors, lower social competence, and greater risk for suicide attempts.

When maternal and paternal NAD were both added to the model, maternal MDD was independently associated with more symptoms of physical illness. Surprisingly, however, maternal NAD was associated with fewer symptoms of physical illness. Paternal MDD continued to be associated with lower GPA, more minor stressors, more major stressors, lower social competence, and greater risk for suicide attempts. Also, paternal NAD was associated with lower GPA.

When offspring psychopathology (MDD, anxiety, SUD, and disruptive behavior disorders) was added to the model, maternal MDD was independently associated with more physical illness, and paternal MDD was associated with more major stressors, lower social competence, and greater risk for suicide attempts. Paternal NAD also continued to be associated with lower GPA.

T₃ outcomes—Data for these analyses are also summarized in Table 1. When controlling for interview status of both mothers and fathers and demographic covariates, maternal MDD was independently associated with lower education attained, less peer support, more minor stressors, lower life satisfaction, and greater mental health utilization (MHU). Paternal MDD was independently associated with lower social competence and greater risk for suicide attempts.

When maternal and paternal NAD were added to the model, maternal MDD continued to be associated with lower education, less peer support, more minor stressors, lower life satisfaction, and greater MHU. Paternal MDD was associated with lower social competence, greater risk for suicide attempts, and greater MHU. Paternal NAD was also associated with lower educational attainment.

When offspring psychopathology was included in the model, maternal MDD was independently associated with more minor stressors and greater MHU, and paternal MDD was associated with lower social competence. Paternal NAD was associated with lower education attained.

Parental MDD X Offspring sex interactions—We also examined whether the association between parental MDD and offspring functioning was moderated by offspring gender. For these analyses, the main effects for maternal and paternal interview status, the demographic covariates, maternal and paternal MDD, and the interaction terms for maternal MDD X offspring sex and paternal MDD X offspring sex were entered simultaneously.

Only 1 of these 46 two-way interactions was significant. Paternal MDD interacted with offspring sex in predicting T₁ school difficulties ($\beta = .28, p < .05$), such that male offspring of depressed fathers had the greatest school difficulties.¹

Discussion

We addressed six questions. First, we examined the relative impact of maternal versus paternal MDD. As noted earlier, the effects of paternal MDD on offspring have not been examined as extensively as the effects of maternal depression, and maternal depression has typically been

found to be more strongly associated with offspring outcomes than paternal MDD. Our findings indicate that paternal MDD plays an important role in offspring psychosocial functioning, particularly in adolescence. Considering our baseline model, we found that paternal MDD was associated with five areas of functioning in adolescent offspring, whereas maternal MDD was not associated with any of the outcome measures. In young adulthood, however, we found that maternal MDD had significant associations with five measures of psychosocial functioning, compared to two areas for paternal MDD. We explored whether the effects of parental MDD operated through exposure to the parent, as indexed by whether offspring were living with the parent at the time of the adolescent assessment (see footnote 1). In these analyses, we found little evidence that the effects were moderated by exposure to a depressed parent.

Second, we investigated whether parental depression is more closely associated with some areas of functioning than others. This appeared to be the case. However, few clear patterns emerged. Indeed, the pattern of associations appeared to be specific not only to the area of functioning, but also to the sex of the depressed parent, and the developmental period of the offspring.

Third, we expected to find more associations between parental psychopathology in adolescence than in young adulthood. This expectation was not supported. During adolescence, we found significant associations for 5 of 10 outcomes, and in young adulthood we found 7 of 13 significant associations. Thus, the effects of parental depression on offspring functioning appear to persist into adulthood. It was also striking that the importance of parental gender appeared to change over time. During adolescence, paternal depression was associated with impairment in multiple domains, whereas during young adulthood, maternal depression was associated with impairment in a greater number of domains.

Fourth, we investigated whether the impairments were uniquely associated with parental depression as opposed to other forms of psychopathology. During adolescence, we found six associations for parental MDD and two for parental NAD when all forms of parental psychopathology were entered into the regression model. During young adulthood, we found seven associations for parental MDD and one for parental NAD. This suggests that many of the effects of parental psychopathology on offspring functioning are specific to parental MDD. However, it should be noted that we did not disaggregate the various forms of parental NAD. It is possible that some forms of parental NAD may have greater associations with offspring functioning than others.

Fifth, we examined whether the psychosocial impairment in offspring of depressed parents can be explained by psychopathology in the offspring themselves. When including offspring psychopathology in the regression model, paternal MDD continued to have a negative impact

¹We conducted two subsidiary sets of analyses. First, we examined whether the associations between parental MDD and offspring functioning differed as a function of exposure to the parent, as indexed by living with the parent at T₁. For these analyses, main effects for maternal and paternal interview status, offspring sex, parental education, living with both biological parents at T₁, maternal and paternal MDD, and interaction terms for maternal MDD X living with biological mother at T₁ and paternal MDD X living with biological father at T₁ were entered simultaneously. Only two of the 46 interaction terms were significant. The association between maternal MDD and suicide attempts at T₁ was moderated by whether the offspring lived with their biological mother at T₁ (OR = 1.59, $p = .05$). Maternal MDD was associated with risk for offspring suicide attempts at T₁ only if the offspring lived with their biological mother. The association between paternal MDD and major stressors at T₃ was moderated by whether the offspring lived with their biological father at T₁ ($\beta = 1.40$, $p < .05$). Paternal MDD was associated with more major stressors at T₃ if the offspring lived with their biological father. Second, we examined whether the associations between parental MDD and offspring functioning were moderated by parental interview status. For these analyses, main effects for maternal and paternal interview status, offspring sex, parental education, living with both biological parents at T₁, maternal and paternal MDD, and interaction terms for maternal MDD X maternal interview status and paternal MDD X paternal interview status were entered simultaneously. Only two of the 46 interaction terms were significant. The association between maternal MDD and family support at T₁ was moderated by maternal interview status ($\beta = .31$, $p < .05$). Maternal MDD was associated with lower family support at T₁ if the biological mother was directly interviewed. The association between maternal MDD and peer support at T₃ was moderated by maternal interview status ($\beta = .31$, $p < .05$). Paternal MDD was associated with lower peer support at T₃ if the biological father was directly interviewed.

on major stress, social competence, and suicidal risk in adolescence and on self-perceived social competence in young adulthood. Additionally, maternal psychopathology had a negative impact on self-reported physical symptoms in adolescence and on minor stress and mental health utilization at young adulthood. Thus, many of the effects of parental MDD on offspring functioning persisted even after controlling for psychopathology in the offspring.

Lastly, we investigated whether offspring gender moderated the associations between parental MDD and offspring outcomes. This did not appear to be the case. For almost all of the outcomes examined, the influence of maternal and paternal MDD were similar regardless of offspring gender.

This study adds to the literature documenting increased rates of MDD and elevated levels of psychosocial impairment in the offspring of depressed parents (Beardslee et al., 1998; Goodman & Gotlib, 2002; Weissman et al., 1997), by demonstrating that offspring of depressed parents exhibited significant psychosocial impairment even in the absence of diagnosable psychopathology. These impairments may have implications for later economic productivity, and interpersonal and family functioning in later adulthood. In addition, they have clinical implications, as psychosocial functioning should be monitored in children of depressed parents, and may provide useful targets for preventative interventions.

In evaluating our findings, several limitations should be considered. First, we did not assess all domains of psychosocial functioning. In the future, we plan to explore the effects of parental psychopathology on marital quality and attainment, and offspring parenting of their own children in later adulthood.

Second, we conducted many analyses, which increases the likelihood of Type 1 errors. However, we emphasized the pattern of findings, rather than any particular finding.

Third, our ability to assess exposure to parental depression was limited. Better measures of exposure to parental MDD are needed. Moreover, further work is needed to determine the mechanisms that underlie the associations between parental MDD and offspring impairment.

Fourth, we were not able to directly interview all of the parents. However, we controlled for the effect of parental interview status in all analyses. In addition, we examined whether parental diagnostic status moderated the association between parental MDD and offspring functioning, and few of these interactions were significant.

Finally, we did not use the same set of outcome measures at T₁ and T₃. Although similar constructs were assessed at the two developmental periods, we were not able to examine the effects of parental MDD on the longitudinal trajectory of offspring functioning.

In conclusion, the findings indicated that paternal, as well as maternal, MDD is associated with impairment in offspring. Second, the associations between parental MDD and offspring impairment were domain-specific and appeared to be dependent on parental gender and the developmental period of the offspring. Third, associations between parental psychopathology and offspring functioning were evident in both adolescence and young adulthood. Fourth, the associations between parental psychopathology and offspring impairment were fairly specific to parental MDD, as opposed to other parental disorders. Finally, many of the associations between parental MDD and offspring psychosocial impairment persisted even after controlling for psychiatric disorders in the offspring, indicating that the effects of parental depression on offspring functioning are not entirely mediated by offspring psychopathology.

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Table 1

Unstandardized Regression Coefficients (Standard Errors) and Odds Ratios (and 95% Confidence Intervals) for the Relationships Between Parental Psychopathology and Offspring Outcomes.

Offspring Outcome Measures	Parental Diagnosis										
	Model 1			Model 2			Model 3				
	M-MDD	P-MDD		M-MDD	P-MDD	M-NAD	P-NAD	M-MDD	P-MDD	M-NAD	P-NAD
T ₁ Continuous Outcomes (Beta, SE)											
GPA	<-.01 (.07)	-.24** (.09)		<-.01 (.07)	-.20* (.09)	.02 (.06)	-.14* (.06)	.02 (.07)	-.16 (.09)	.02 (.06)	-.13* (.06)
School Problems	.06 (.05)	.06 (.06)		.06 (.06)	.06 (.07)	<.01 (.05)	-.03 (.05)	.04 (.06)	.02 (.06)	.01 (.05)	-.04 (.05)
Family Support	.11 (.06)	.11 (.06)		.11 (.06)	.09 (.06)	.01 (.05)	.07 (.05)	.09 (.06)	.03 (.06)	.02 (.05)	-.06 (.05)
Peer Support	<-.01 (.04)	.03 (.05)		<.01 (.04)	.03 (.05)	<-.05 (.04)	<-.01 (.04)	<-.01 (.04)	<-.01 (.05)	<-.05 (.04)	<-.01 (.05)
Minor stressors	1.05 (1.15)	2.83* (1.26)		.67 (1.16)	2.93* (1.31)	1.76 (1.05)	-.32 (1.03)	.09 (1.12)	1.80 (1.30)	1.94 (1.03)	-.67 (1.02)
Major stressors	.06 (.06)	.31*** (.07)		.07 (.07)	.29*** (.07)	-.04 (.06)	.05 (.06)	.06 (.06)	.27*** (.07)	-.04 (.06)	-.04 (.06)
Social competence	.04 (.06)	-.20* (.08)		.04 (.06)	-.20* (.08)	.02 (.06)	.02 (.06)	.05 (.06)	-.17* (.08)	.01 (.06)	-.04 (.05)
Physical illness	.13 (.07)	.15 (.09)		.16* (.07)	.12 (.10)	-.11* (.06)	.08 (.06)	.14* (.07)	.09 (.10)	-.10 (.05)	-.06 (.05)
T ₁ Dichotomous Outcomes (OR, 95% CI)											
Suicide attempt	1.48 (.78–2.80)	3.28*** (1.64–6.52)		1.71 (.89–3.27)	3.31** (1.64–6.67)	.53 (.27–1.05)	.95 (.53–1.73)	1.36 (.71–2.64)	2.65* (1.19–5.92)	.58 (.29–1.15)	-.80 (.42–1.53)
MHU	1.33 (.83–2.11)	1.29 (.75–2.22)		1.34 (.81–2.19)	1.33 (.76–2.33)	.94 (.57–1.57)	.89 (.56–1.42)	1.08 (.61–1.93)	.80 (.41–1.59)	.98 (.56–1.74)	-.73 (.44–1.22)
T ₃ Continuous Outcomes (Beta, SE)											
Education	-.34* (.17)	-.25 (.19)		-.37* (.17)	-.12 (.19)	.06 (.15)	-.42** (.15)	-.27 (.17)	-.02 (.18)	.04 (.15)	-.35* (.14)
Income	-.09 (.13)	.27 (.18)		-.06 (.14)	.29 (.19)	-.14 (.14)	-.07 (.13)	-.03 (.14)	.30 (.19)	-.15 (.13)	-.03 (.12)
Unemployment	-.02 (.02)	.02 (.03)		-.02 (.02)	.01 (.03)	<.01 (.02)	<.04 (.02)	-.03 (.02)	<.01 (.03)	<.01 (.02)	.03 (.02)
Family Support	-.28 (.25)	-.05 (.29)		-.22 (.26)	-.02 (.31)	-.29 (.24)	-.13 (.22)	-.11 (.26)	.08 (.30)	-.31 (.24)	-.04 (.21)
Peer support	-.36* (.17)	-.06 (.20)		-.35* (.17)	-.03 (.20)	-.10 (.16)	-.26 (.15)	-.29 (.17)	.06 (.20)	.09 (.16)	-.24 (.16)
Minor stressors	3.43** (1.06)	.58 (1.29)		3.29** (1.08)	.72 (1.31)	.61 (1.01)	.47 (.95)	2.52* (1.07)	.24 (1.29)	.64 (1.00)	-.105 (.92)
Major Stressors	.19 (.29)	.21 (.35)		.25 (.29)	.16 (.36)	-.21 (.25)	.15 (.25)	.02 (.26)	.04 (.34)	-.19 (.24)	-.02 (.24)
Social Competence	.17 (.44)	-.125* (.49)		.17 (.44)	-.132 (.50)*	.02 (.40)	.23 (.37)	.34 (.44)	-.121* (.49)	.02 (.39)	.30 (.38)
Physical Illness	.04 (.06)	.01 (.06)		.04 (.06)	.03 (.06)	<.01 (.05)	-.05 (.05)	<-.01 (.06)	<.01 (.06)	<.01 (.05)	-.09 (.05)
Life Satisfaction	1.52* (.76)	1.22 (.89)		1.60* (.77)	1.42 (.90)	-.48 (.72)	.67 (.66)	.97 (.76)	1.01 (.85)	-.47 (.72)	-.99 (.64)

Offspring Outcome Measures	Parental Diagnosis					
	Model 1		Model 2		Model 3	
	M-MDD	P-MDD	M-MDD	P-MDD	M-MDD	P-MDD
T ₃ Dichotomous Outcomes (OR, 95% CI)						
Suicide attempt	1.78 (.72–4.38)	2.84* (1.09–7.41)	1.79 (.70–4.60)	2.88* (1.08–7.70)	1.25 (.49–3.19)	2.51 (.93–6.75)
MHU	2.29*** (1.47–3.59)	1.66 (.97–2.84)	2.18** (1.38–3.46)	1.75* (1.02–3.00)	1.86* (1.14–3.03)	1.61 (.91–2.85)
Live with parent	.99 (.59–1.66)	1.14 (.62–2.09)	1.00 (.58–1.72)	1.17 (.63–2.18)	1.08 (.63–1.88)	1.34 (.72–2.52)

* p<.05;
 ** p<.01;
 *** p<.001

Note. For each model parental diagnoses are simultaneously entered. All models are adjusted for maternal interview status, paternal interview status, offspring gender, living with both biological parents as an adolescent, and parental education. Model 3 is also adjusted for offspring lifetime diagnosis of major depressive disorder, anxiety disorder, substance abuse or dependency disorder, and disruptive behavior disorders. Parental diagnosis are simultaneously entered. M- = maternal, P- = paternal. MDD = major depressive disorder, NAD = non-affective disorder, SE = standard error, OR = odds ratio.