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The Influence of Paternal and Maternal Major Depressive Disorder on Offspring Psychiatric Disorders

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Introduction

One of the largest known risk factors for the development of depression among youth is a family history (for a review see Beardslee, Gladstone, & O'Connor, 2011). At least 15 million children live with a depressed parent (England & Sim, 2009) and the number of children exposed to parental depression is likely larger when considering the developmental span of childhood through adolescence. Adverse family environments are one of the most consistent identified risk factors for adolescent depression (Evans et al., 2005) and the importance of familial factors may contribute to depression among youth even more than the influence of peers (Sheeber, Hops, & Davis, 2001).

High rates of depression among offspring of depressed mothers have been well documented (Goodman & Gotlib, 1999; Halligan, Murray, Martins, & Cooper, 2007; Hammen & Brennan, 2003) with some studies suggesting depression in offspring is more closely related to depression in mothers than to depression in fathers (Connell & Goodman, 2002). In contrast, ecological models of developmental psychopathology suggest that parental influence on the child is not limited to mothers (e.g., Cicchetti & Toth, 1997). Yet, few studies examine the influence of paternal depression on offspring outcomes (Kane & Garber, 2004, 2009). A review published in 2005 found that only 25% of developmental psychopathology research examined data from mothers and fathers in predicting offspring outcomes (Phares, Fields, Kamboukos, & Lopez, 2005).

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The few studies that have examined the role of a father's mental health on their offspring have documented small effect sizes. A recent meta-analysis documented an effect size of .24 for paternal depression on offspring mental health (Kane & Garber, 2004). This effect is considered small according to guidelines (Cohen, 1988; .2 = small, .5 = medium, .8 = large), but was larger than a previously documented effect size of .14 (compared to .18 for maternal depression; Connell & Goodman, 2002).

A recent study documented the influence of paternal depression on the mental health of offspring in a dataset of 22,000 children (Weitzman, Rosenthal, & Liu, 2011). Parental mental health problems and offspring emotional and behavioral problems were measured via parent self-report of their own mental health as well as their child's. Paternal mental health problems were associated with a 33 – 70% increased risk of emotional or behavioral problems among offspring, whereas maternal mental health problems were associated with a 50 – 350% increased risk when compared to offspring of parents without mental health problems. This recent and large study in particular suggests that the effect of paternal depression may be larger than expected, particularly when adequate samples can be captured.

Exploration of the differential influence of paternal and maternal depression is warranted for theoretical reasons as well. For example, known mechanisms influencing the intergenerational transmission of depression such as genetic, environmental, and gene-by-environment pathways may differ based on whether the affected parent is the father or mother of the child. Parent-child interactions are one hypothesized mechanism in the intergenerational transmission of depression and mothers with a history of depression demonstrate high levels of criticism and emotional enmeshment with their offspring (Tompson et al., 2011). Fathers' parenting abilities are also influenced by depression, but less research has explored these mechanisms. Father-child conflict has been found to mediate the contribution of paternal depression on offspring's internalizing and externalizing symptoms (Kane & Garber, 2009). Depressed fathers' expressions of positive affect and verbal interactions with offspring have been found to be negatively related to offspring internalizing symptoms (Jacob & Johnson, 1997). In this same study, paternal expressions of approval were inversely related to offspring depressive symptoms. Thus, what is clear is that both paternal and maternal depression influence one's ability to effectively parent. However, the difference in roles of mothers and fathers may lead to a differential effect in the development of psychopathology among offspring. Differences in parent-child interactions between fathers and mothers offer just one theoretical rationale for including both parents when examining the intergenerational transmission of depression. These theoretical arguments for a differential role of fathers and mothers in offspring psychopathology provides a foundation for the current exploration.

In addition, paternal depression and maternal depression may differentially influence the timing of psychopathology onset among offspring. Longitudinal twin data from 670 twin pairs ages 5–17 found that shared environmental effects more significantly influenced levels of depression among younger children (Scourfield et al., 2003), whereas heritability estimates rose during adolescence. Thus, the mechanisms of transmission may differ

between fathers and mothers and these mechanisms may differentially influence the timing of disorder onset among offspring.

We sought to contribute to the growing literature by examining the effect of paternal and maternal depression on offspring using a large sample of offspring carefully assessed with a clinical diagnostic interview conducted by a clinician blind to parental depression status within a longitudinal high risk study. Given the burden of the evidence, we hypothesized that maternal depression would more greatly influence offspring mental health (MDD, anxiety, and substance use disorders). We also examined level of offspring functioning as one way of measuring the influence of paternal and maternal depression. In addition, we conducted exploratory analyses of the effect of paternal and maternal depression on age of disorder onset. Our hypothesis was that maternal depression would influence young children more given the significance of the mother in the early rearing environment.

Method

Participants

The study design is a retrospective, longitudinal, and multigenerational cohort. Hypotheses were tested in a sample followed clinically over 25 years that included five waves of assessments between 1982 (Wave 1) and 2010 (Wave 5). The sample now includes three generations: (grandparents (G1), parents (G2), and grandchildren (G3)). The design includes families at high and low risk for depression based on the depression status of the original sample (G1). The focus of the current investigation was G2 who participated in Wave 1 and includes clinical follow-up data collected in Waves 2 through 5 to examine the influence of paternal and maternal MDD on lifetime diagnoses as offspring age. The focus was on G2, instead of G3, to include some representation of adult onset diagnoses. Two hundred and twenty biological offspring (ages 6 – 23, mean age = 17.0 at the time of the Wave 1 assessment) from the G2 cohort participated. Fifty-two percent were female, all participants were white and families were on average working/middle class.

The G1 sample derives from the Yale Family Study of Depression (Weissman, Leckman, Merikangas, Gammon, & Prusoff, 1984). Of the eligible families (n=105) with children between 6 and 23 years of age at the time of the Wave 1 assessment, 87.5 % agreed to participate and those who refused were divided equally between depressed and non-depressed families, suggesting recruitment was not biased toward one of the risk groups. One or more of the parents from the depressed families had received treatment for depression. The low risk, non-depressed families derived from a community study in New Haven, Connecticut (Weissman & Myers, 1978) and reported no history of treatment or psychiatric illness. The overall sample has been described in detail elsewhere (Weissman, Fendrich, Warner, & Wickramaratne, 1992; Weissman et al., 1987; Weissman, Warner, Wickramaratne, Moreau, & Olfson, 1997; Weissman et al., 2005).

Procedures

Mothers completed information about prenatal, birth, and postnatal events for each of their children. Children were not directly interviewed about their mental health until the age of 6.

Diagnostic assessments of G2 offspring were reviewed by a psychiatrist or psychologist who was blind to G1 diagnosis and previous G2 diagnostic assessments using best estimate procedures (Leckman, Sholomskas, Thompson, Belanger, & Weissman, 1982; Weissman et al., 2005). Each case was reviewed independently by a second blind rater. In the case of a disagreement, a consensus diagnosis was made again, always blind to the initial diagnosis. For additional details on the sample, assessments, training, inter-rater reliability, and monitoring for quality control see previous publications (Leckman, Sholomskas, Thompson, Belanger, & Weissman, 1982; Weissman et al., 2005).

Measures

The Kiddie-Schedule for Affective Disorders and Schizophrenia Epidemiologic or Present and Lifetime version (KSADS-PL; Kaufman, Birmaher, Brent, Ryan & Rao, 2000) was used for subjects under the age of 18. The Schedule for Affective Disorders and Schizophrenia Lifetime version (SADS-LA; Mannuzza, Fyer, Klein, & Endicott, 1986) was used for all subjects 18 years of age and older. The Global Assessment Scale (GAS; Endicott, Spitzer, Fleiss & Cohen, 1976) and the child version (C-GAS; Shaffer et al., 1983) were also completed. GAS and C-GAS ratings are based on a 0–100 point scale providing an overall estimate of a person's current functional adjustment based on all available information. Lower scores indicate more overall impairment in functioning.

G1 depression was defined according to Research Diagnostic Criteria (RDC; Weissman, Fendrich, Warner, & Wickramaratne, 1992; Weissman, Gammon, John, Merikangas, Warner, & Prusoff, 1987; Weissman, Warner, Wickramaratne, Moreau, & Olfson, 1997; Weissman, Wickramaratne, Nomura, Warner, Verdeli, & Pilowsky, 2005) modified to require 4 weeks duration and impairment. Lifetime DSM-IV MDD was used as the main outcome for G2. G2 any anxiety disorder included all childhood and adult lifetime DSM-IV anxiety disorders, such as Generalized Anxiety Disorder and Social Phobia, Specific Phobia, Agoraphobia, and Panic Disorder. G2 substance use included lifetime DSM-IV Alcohol Abuse and Dependence, as well as Substance Abuse and Dependence.

Data Analyses

Group differences in demographic variables were tested using t-tests for continuous data and chi-square tests for categorical data. Relative risk of offspring diagnoses were calculated in SAS (version 9.0, SAS institute Inc., USA) controlling for offspring age and correlation within the family as well as the presence of disorder in the alternate parent. Cox proportional hazards models were used instead of logistic regression to adjust for differential length of follow-up (Cox, 1972). Correlated outcomes within family were adjusted using the approach of Lin & Wei (Lin & Wei, 1989). To specifically compare the effects of maternal MDD and paternal MDD on rates of offspring MDD, a separate model was run including both of these terms and their interaction.

To assess variation in the effect of paternal and maternal depression on age of onset, three separate Cox proportional hazard models (Cox, 1972; Cox & Oakes, 1984) were fitted, wherein the incidence of risk for MDD, any anxiety, and substance use were calculated on the following age groups: < 13 years old, 13–18 years old, and greater than 18 years old. We

chose this specific method as it takes into account differential length of follow-up. We used these categories to correspond with pre-adolescent onset, adolescent onset, and adult onset of disorder. Mixed models were used to examine global functioning outcomes in which family was treated as a random variable. The interaction of maternal and paternal MDD was included to measure the interactive effect of having two parents with MDD on offspring mental health outcomes. Last, to rule out the possibility that findings derived from confounders such as parental comorbidities including substance use, anxiety disorders, and parental living arrangement (i.e., presence in the home), we re-ran models including these terms.

Results

Thirty seven offspring had both a mother and father with a history of depression, whereas one hundred seventeen had a mother with depression and seventy three had a father with depression. Sixty seven offspring did not have a parent with depression. Demographics of the groups (Father MDD, Mother MDD, and both MDD) were similar. There were no demographic differences in offspring gender, age, or socio-economic status by parental MDD status. G2 offspring of mothers with MDD as compared to mothers without MDD were slightly younger at the Wave 1 assessment; in 9^{th} as compared to 10^{th} grade (t = 2.2, p=.03). No statistically significant demographic differences were detected by paternal MDD. One hundred and twenty seven offspring (58%) were living with both biological parents at Wave 1, an additional 45 (20%) lived with their biological mother, but not father, and six (<1%) lived with their biological father and stepmother. Forty two (19%) lived with neither biological parent due to living at college or moving away from home during early adulthood. There were no group differences in number of marriages or current marital status. At Wave 1–22% (n = 18) of parents had divorced. Five mothers and three fathers remarried.

Rates of MDD and anxiety were significantly higher among offspring of depressed fathers and mothers respectively, when compared to non-depressed fathers and mothers (Table 1). Both paternal (RR = 3.1, 95% CI = 1.6, 6.1, $p < .01$) and maternal (RR = 2.7, 95% CI = 1.5, 5.0, $p < .01$) depression significantly increased the prevalence of MDD among offspring. The interaction in this model was also significant (RR = .33, 95% CI = .12, .89, p = .03). Maternal depression led to significantly lower overall functioning, whereas paternal depression did not. In contrast, offspring of two depressed parents had slightly higher functioning than offspring in the other groups. In terms of potential confounds, comorbid substance use and parental presence in the home did not significantly contribute to statistical models. The inclusion of comorbid maternal anxiety reduced the influence of maternal MDD on offspring anxiety resulting in a non-significant influence of maternal MDD on offspring global functioning.

In terms of age of onset, paternal MDD (RR = 14.8, 95%, CI = 1.8, 21.8, $p < .01$), but not maternal MDD (RR = 4.1, 95% CI = .77, 21.9, $p = .09$) was associated with anxiety disorder onset occurring during adulthood (after age 18; $\beta = 17.4$, $p < .01$). In contrast, maternal MDD (RR = 2.8, 95% CI = 1.2, 56.4, $p = .02$), but not paternal MDD (RR = 1.2, 95%, CI = .55, 2.7, $p > .05$), was associated with MDD onset among offspring in childhood (prior to age 13; $\beta = -2.0$, $p = .03$).

Discussion

Our findings indicate that when offspring outcomes are assessed via diagnostic interview depression in fathers is associated with increased rates of depression and anxiety when compared to non-depressed fathers. This parallels the increased rates of depression and anxiety observed among offspring of depressed mothers compared to non-depressed mothers. In other words, *both* fathers and mothers matter in the intergenerational transmission of depression. This was in contrast to our specific hypothesis that maternal depression would more greatly influence offspring, but in line with more recent studies that have included measures of paternal depression.

Maternal depression resulted in lower levels of global functioning among offspring, which was in line with our hypothesis. Specifically, offspring of depressed mothers compared to non-depressed mothers were more likely to fall into a slightly lower functioning category. For example, global functioning in the 61–70 range indicates “some difficulty” in one domain of life (i.e., school, family, relationships with peers). This finding is consistent with the previous literature (e.g., Rohde, Lewinsohn, Klein, & Seeley, 2005) and may be related to the association of maternal MDD in depression onset earlier in life.

In fact, maternal MDD resulted in offspring disorder onset prior to age 13 which may place children on a negative trajectory resulting in poorer levels of functioning as they age. Another possibility is that mothers have a larger, or more frequently observed and measured, role in their offspring’s early rearing. Indeed, much of the extant research has focused on the influence of maternal stress during pregnancy or in early childhood on offspring outcomes (Ho, Burggren, 2010; Oberlander et al., 2008). A promising future research direction involves the examination of whether fathers exacerbate the effects of maternal depression on offspring psychopathology through genetic or environmental mechanisms (Goodman & Gotlib, 1999).

Surprisingly, there was an interaction of maternal and paternal depression on offspring functioning such that these offspring were functioning slightly higher than offspring of one depressed parent (in the 71–80 range reflecting “no more than slight” impairment). This may be due to low power to detect an interaction within this smaller group. It is also possible that families in which two parents had MDD may have been more likely to rely on their extended family, friends, and their communities to effectively raise their child.

Comorbid maternal anxiety decreased the influence of maternal MDD on offspring MDD. Specifically, inclusion of comorbid anxiety rendered the relation between maternal depression and offspring global functioning non-significant. It is possible that comorbid anxiety partially dilutes the influence of maternal MDD on offspring functioning or that this comorbid phenotype influences offspring in domains other than global functioning as measured by an independent evaluator. Future research on the effects of maternal comorbidity on child developmental outcomes is certainly warranted.

Last, paternal MDD resulted in anxiety onset among offspring after age 18. We are not aware of previous findings indicating that paternal depression influences the onset of anxiety disorders in offspring older than 18 and believe this is an important future direction for

future research. It is critical to include fathers in research of these mechanisms to better understand these nuanced results. The relative dearth in studies examining the effect of paternal depression on offspring may stem from several factors. Possibilities include the lower prevalence of depression among males and their lower rates of treatment seeking behavior (Wang et al., 2005).

As noted, the current study has limitations including the modest sample size. This limitation is contrasted with the strength of highly detailed and reliable diagnostic information. We framed our analyses of developmental phase during age of disorder onset as exploratory in light of the sample size. In addition, findings may not generalize to a more mildly ill population or other ethnic groups as we examined a White cohort deriving from moderate to severe depression in the G1 proband. Of note, a meta-analysis found a greater association of paternal depression with internalizing psychopathology in children in community samples, compared to clinical samples (Kane & Garber, 2004). These authors discuss possibilities for the smaller effects of paternal depression on offspring in clinical (as opposed to community) samples as possibly stemming from restriction of range or a threshold effect. Thus, the current results may be due, in part, to the clinical nature of the high risk group. Last, future research should more closely examine the exact timing of parental MDD onset and duration on child development outcomes. More detailed research examining the timing and duration of exposure to parental depression during early development is critically important to understanding the mechanisms of risk.

In sum, our finding indicating that paternal depression influences offspring just as much as maternal depression is in line with meta-analytic results (Wilson & Durbin, 2010) and suggests that the influence of paternal depression warrants at least as much attention as that of maternal depression. This is particularly important for research on prevention and intervention. Familial interventions that include both parents may offer the best mechanism for the prevention of mental health disorders among offspring. Indeed, a randomized trial of two public health prevention interventions for parental depression demonstrated that both interventions produced sustained effects through 4–5 years post-intervention (Beardslee et al., 2007). Families randomized to the clinician-based intervention had significantly more gains in parent-child behaviors and attitudes and in child-reported understanding of their parent’s disorder. Continued research on how to engage fathers in treatment of their own depression and in prevention of transmission to their offspring is warranted and this may be best accomplished by focusing on the family as a whole. Moreover, in light of our finding that maternal depression resulted in depression onset among offspring during childhood we recommend prevention efforts be delivered no later than middle childhood. Evidence suggests that treatment of maternal depression ameliorates some of the burden of psychopathology experienced by offspring (Weissman et al., 2006; Weissman et al., in press).

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Table 1

Offspring MDD, anxiety, and substance use by paternal and maternal MDD controlling for age and correlation within family

	Father – no MDD	**Father - MDD**		**Mother – no MDD**	**Mother – MDD**		**Interaction Mother*Father**
	N = 147	**N = 73**		**N = 103**	**N = 117**		**N = 37**
	N(%)	**N(%)**	**RR (CI 95%)**	**N(%)**	**N(%)**	**RR (CI 95%)**	
Lifetime anxiety	56(38.1)	46(63.0)	1.9(1.3,3.1)*	33(32.0)	69(58.9)	2.3(1.4,3.6)*	n.s.
Lifetime MDD	66(44.9)	43(58.9)	1.6(1.1,2.5)*	40(38.8)	69(58.9)	2.1(1.3,3.2)*	n.s.
Lifetime substance use	52(35.4)	20(27.4)	.62(.35,1.1)	34(33.0)	38(32.5)	1.1(.74,1.7)	n.s.
Global Assessment Scale (GAS)	Mean(S.E.) 68.5(1.4)	Mean(S.E.) 66.8(2.0)	β = 1.7	Mean(S.E.) 71.8(1.7)	Mean(S.E.) 63.5(1.6)	β = 8.5*	β = −9.8* 73.0(3.8)

Note. MDD = Major Depressive Disorder; S.E. = standard error; RR = Relative Risk; CI = Confidence Interval

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