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Research paper

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.

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.

1. Introduction

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 and 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 and 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 and 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 and 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. 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 and 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 et al., 2000; Knafl et al., 2015). Following Goodman and Gotlib's

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(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 and 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 and 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 et al. (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 et al. (2005) found a measure of family conflict to mediate the link between mother's lifetime depression severity and children's behavioral problems; Bifulco et al. (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 and 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 specifically (Garber and 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 and Kazdin, 2005). Indeed, perspectives of different family members vary, with offspring tending to adopt more negative perspectives on family functioning than their parents (Noller and Callan, 1986; Tamplin and 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.

2. Methods

2.1. 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 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.

2.2. Recruitment and procedure

All mothers had participated in a previous Program Project during