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Long-term Effects of Fathers' Depressed Mood on Youth Internalizing Symptoms in Early Adulthood

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Abstract

While an accumulating body of research has documented increased risk for psychopathology among children of depressed fathers, most studies have used cross-sectional design and little is known about offspring outcomes beyond childhood. Using prospective data from a community sample (N = 395), we found that paternal depressive symptoms when children were in early adolescence (age 13) predicted offspring depressive and anxiety symptoms at age 21, controlling for baseline youth symptoms, maternal depressive symptoms, and other known correlates of internalizing problems in early adulthood. Associations were not moderated by maternal depressive symptoms or child gender. These results suggest that the unique and long-term effects of paternal depression on children's risk for mood disorders may persist into adulthood.

Children of depressed parents are at increased risk for psychiatric disorders and developmental difficulties (for reviews, see Beardslee, Versage, & Gladstone, 1998; Downey & Coyne, 1990; Goodman & Gotlib, 2002; Hammen, 2008). A wealth of research has documented significant associations between maternal depression and children's risk for impaired cognitive functioning, major depression, clinical levels of depressive and anxiety symptoms, and behavioral problems (for meta-analysis, see Goodman et al., 2011). Much less attention has been directed toward the adverse effects of paternal depression on children's psychological health (Phares, 1996; Phares, Duhig, & Watkins, 2002). To illustrate, in a review of 514 articles related to child and parent psychopathology published

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between 1992 and 2005, 45% included mothers only, 2% included fathers only, 25% included both mothers and fathers and analyzed the data separately, and 28% included both mothers and fathers but either failed to indicate parent gender or did not analyze the data separately (Phares, Fields, Kamboukos, & Lopez, 2005). The lack of research on children of depressed fathers is likely due to a number of factors, including the higher prevalence of depression in women, the widespread assumption that fathers are relatively unimportant to the social-emotional development of their children, and a historical tendency to attribute children's psychological problems to mothers rather than fathers (Phares, Lopez, Fields, Kamboukos, & Duhig, 2005). Another possible reason for the disproportionate focus on maternal versus paternal depression is that fathers may be more difficult to recruit for research on child development (e.g., Cassano, Adrian, Veits, & Zeman, 2006).

Recent evidence indicates that fathers have more influence on their children than previously thought (Jaffee, Moffitt, Caspi, & Taylor, 2003). For example, social scientists have begun to recognize the importance of paternal depression, both in terms of prevalence and in relation to children's psychological functioning (for recent reviews, see Bradley & Slade, 2011; LeFrancois, 2012; Paulson & Bazemore, 2010; Ramchandani & Psychogiou, 2009). Studies have shown that depression rates are not significantly different for mothers and fathers during pregnancy (Field, Diego, & Hernandez-Reif, 2006) and that an estimated 21% of fathers experience at least one episode of major depression by the time children reach 12 years of age (Davé, Petersen, Sherr, & Nazareth, 2010). A growing body of work has demonstrated that, beginning in infancy and continuing throughout childhood, offspring of depressed fathers are at elevated risk for a number of psychiatric problems (e.g., Field, Diego, & Hernandez-Reif, 2010; Greene, Pugh, & Roberts, 2008; Gross, Shaw, Moilanen, Dishion, & Wilson, 2008; Manning & Gregoire, 2006; Ramchandani et al., 2008, Ramchandani, Stein, Evans, & O'Connor, 2005; Spector, 2006; Weitzman, Rosenthal, & Liu, 2011). Two meta-analyses (Connell & Goodman 2002; Kane & Garber, 2004) have associated paternal depression with children's risk for psychopathology; some evidence suggests comparable effects of maternal and paternal depression on offspring outcomes (e.g., Jacob & Johnson, 1997; Marchand & Hock, 1998). Less is known about how—or under which circumstances—depression in fathers conveys risk to children.

Previous research on children of depressed fathers has been limited by several factors. For example, the majority of studies have relied on cross-sectional data, thus precluding inferences about causation and direction of effect. Of the few existing longitudinal studies, most have not controlled for initial levels of child psychopathology, thereby limiting conclusions about paternal depression as a risk factor for children's maladjustment. Another methodological limitation of this literature has been a failure take into account characteristics of the family environment that could confound statistical relationships between paternal depression and poor child outcomes. For example, factors such as marital discord and economic disadvantage are related to mental illness in parents and children (Flouri, Mavroveli, & Tzavidis, 2010; Hops & Sherman, 2013; Reising et al., 2013); unless these variables are controlled for, it is difficult to know whether offspring risk is a consequence of paternal depression or whether the association between father and child functioning is spurious and actually caused by a third variable. Likewise, with several notable exceptions (e.g., Kane & Garber, 2009; Ramchandani et al., 2005; Weitzman et al.,

2011), little attention has been given to whether depression in fathers is a unique predictor of children's adjustment or whether associations can be explained by the effects of maternal depression.

It is well-established that depressive and anxiety disorders are highly recurrent conditions that often emerge in adolescence or early adulthood (Abela & Hankins, 2009; Costello, Foley, & Angold, 2006; Fergusson, Horwood, Ridder, & Beautrais, 2005; Merikangas et al., 2010; Polanco-Roman & Miranda, 2012; Reinke, Eddy, Dishion, & Reid, 2012). Despite this, research to date on paternal depression and children's risk for internalizing problems has focused primarily on infancy and childhood, to the exclusion of risk transmission in adolescence and beyond—perhaps because of the crucial roles parents play in young children's cognitive and socioemotional development (see Stack & Poulin-Dubois, 1998). To illustrate, the average age of children in studies reviewed for meta-analyses by Connell and Goodman (2002) and Kane and Garber (2004) was 9.5 and 10.7 years, respectively. Of the limited available data using older samples, several studies have reported increased rates of depression and anxiety among adolescent offspring of depressed fathers (e.g., Beardslee, Gladstone, Wright, & Cooper 2003; Brennan, Hammen, Katz, & Le Brocque, 2002; Kane & Garber, 2009; Klein, Lewinsohn, Rohde, Seeley, & Olino, 2004; Reeb & Conger, 2010). In an investigation of parental depression and trajectories of psychopathology in previously depressed adolescents, Rohde, Lewinsohn, Klein, and Seeley (2005) found that depression in fathers was associated with offspring risk for psychosocial impairment—but not depression—in early adulthood. Of the limited research on parental depression and youth outcomes in adulthood, most studies have relied on offspring-report of parent psychopathology, thus leaving open the possibility of shared method variance (see Lorenz, Conger, Simons, Whitbeck, & Elder, 1991) and biased recall, especially among reporters who were currently depressed (Patten, 2003).

In addition to the scarcity of longitudinal data and failure to examine the unique effects of paternal depression beyond childhood, little attention has been given to factors associated with better or worse offspring outcomes. One possible moderator of risk transmission involves assortative mating, or the tendency for parents to be more similar to each other phenotypically than would be expected if they were mated at random (Merikangas, 1982). Compared to controls, depressed individuals are more likely to have a spouse with depression (for meta-analysis, see Paulson & Bazemore, 2010), and some studies have shown that the adverse effects of paternal depression on youth adjustment were exacerbated by the presence of maternal depression (e.g., Brennan et al., 2002; Kahn, Brandt, & Whitaker, 2004). Child gender has also been proposed to moderate the effects of parental depression on children's mental health, although findings have been equivocal (for review, see Sheeber, Davis, & Hops, 2002). Some results suggest that boys may be at higher risk for psychopathology as a consequence of their fathers' depression (Eberhart, Shih, Hammen, & Brennan, 2006; Ramchandani et al., 2005), while other evidence indicates increased vulnerability in girls, particularly among daughters reporting high father hostility (Reeb, Conger, & Wu, 2010).

The central aim of the present study was to extend longitudinal research on children of depressed fathers into early adulthood. Using prospective data from a community sample,

we examined the long-term effects of paternal depressive symptoms when children were in adolescence (age 13) on offspring depressive and anxiety symptoms at age 21. In order to account for the auto-contingency of offspring internalizing symptoms over time, children's baseline symptoms were included as statistical controls in all models. Because research has associated impaired family relationships, financial stress, and maternal depression with young adults' risk for internalizing problems (e.g., Wickrama, Conger, Surjadi, & Lorenz, 2010), our analyses also adjusted for the effects of hostile parenting, marital discord, economic hardship, and maternal depressive symptoms. To minimize biases produced by single sources of information (Podsakoff, MacKenzie, & Podsakoff, 2012), trained observer and multiple family member reports of study variables were used. Based on the preceding literature review, we hypothesized that paternal depressive symptoms would predict offspring risk for depressive (H_1) and anxiety symptoms (H_2) in early adulthood, and that effects would be exacerbated in the context of high levels of maternal depressive symptoms (H_3). Youth gender was also examined as a moderator of the association between father and child psychopathology (H_4).

Method

Participants and Procedure

Our study sample was drawn from the Family Transitions Project (Conger & Conger, 2002), which has followed a community sample of young adolescents from two-parent families into adulthood. Due to the ethnic composition of the area at the study inception (approximately 1% minority families), all participants were White. At Time 1 of the current study (T1; 1989), the sample included complete data for 430 families, each with a child in the 7th grade (*M* age = 12.61 years, *SD* = .55). At Time 2 (T2; 1997), youth were between the ages of 20 and 22 years (*M* = 20.66, *SD* = .55).

The median yearly income for families in the current analyses was $35,300 at T1, about $3,500 less than married couples with children in the United States as a whole. Family per capita income from all sources (e.g., wages, interest, business profits, etc.) averaged $8,120, and total family income ranged from a net loss of $10,200 to $51,800. Ten percent of the families in our sample had incomes below the U.S. Department of Health and Human Services poverty line ($12,100 for four-person families). The average age of fathers and mothers at T1 was 40 and 38, respectively. Both parents averaged 13 years of schooling. After listwise deletion of missing data, the final sample for the current analyses included 395 families (215 females, 180 males; 92% of the original sample). Compared to those with complete data, families not included in the analyses did not significantly differ on any study measure.

Participants were recruited through 34 schools in eight rural Iowa counties. Seventy-eight percent of the eligible families (those containing a 7th grader and her/his two biological parents) agreed to participate. Each year, participating families were visited by a professional interviewer on two occasions. During the first visit, family members completed a set of questionnaires related to physical and mental health, family processes, and economic circumstances. During the second visit, families were videotaped as they engaged in a structured interaction task described in the Measures section.

Measures

Internalizing symptoms—Each family member reported on his or her psychiatric symptoms using subscales of the Symptom Checklist-90-Revised (*SCL-90-R*; Derogatis & Unger, 2010), a multidimensional symptom inventory that assesses psychopathology on a continuous spectrum and has demonstrated reliability and validity (e.g., Bonicatto, Dew, Soria, & Seghezzo, 1997). The *SCL-90-R* Depression Scale (α = .87 for fathers; α = .86 for mothers; α = .87 for offspring at T1, α = .92 at T2) is a 13-item index measuring the degree to which respondents experience symptoms of depression, such as feeling hopeless about the future, thoughts of ending their life, feeling lonely, and crying easily. One item indicating loss of sexual interest was not included in the assessment of adolescent depressive symptoms at age 13. The *SCL-90-R* Anxiety Scale (α = .83 for offspring at T1; α = .89 at T2) includes 10 items measuring symptoms such as nervousness, tension and trembling, panic attacks, and feelings of terror. Each item of the *SCL-90-R* Depression and Anxiety Scales is scored using a 5-point scale (1 = *not at all*, 5 = *extremely*). Items were averaged into separate composite variables for depression and anxiety; higher scores indicate higher levels of symptomatology.

Observed parental hostility—At T1, family members were videotaped as they engaged in a 15-minute structured interaction task during which they sat at a table together and discussed issues and disagreements they had cited as most problematic in a questionnaire they completed earlier in the visit (e.g., parental expectations of children, household chores, important family events). Videotapes were used to examine the quality of behavioral exchanges between family members and were coded by independent observers who had received 200 hours of training on rating family interactions. Hostile parenting behaviors for mothers and fathers were assessed using the Iowa Family Interaction Rating Scale (IFIRS; Melby & Conger, 2001). Using a scale from 1 (*the behavior is not at all characteristic*) to 9 (*the behavior is highly characteristic*), observers rated each parents' hostile, angry, critical, disapproving, rejecting, and/or contemptuous behavior directed toward their adolescent offspring. Interrater reliability estimates for the observations of parent hostility were computed using Intraclass Correlation Coefficient (ICC) procedures (see Choukalas, Melby, & Lorenz, 2000). The average ICCs for mother and father hostility were .29 and .66, respectively.

Marital instability—Marital instability was measured at T1 using the 5-item short form of the Marital Instability Index (Booth, Johnson, & Edwards, 1983), which has been found to effectively discriminate between couples at high versus low risk for divorce. Mothers and fathers each independently rated feelings (e.g., "has the thought of getting a divorce or separation crossed your mind?") and behaviors (e.g., "have you discussed divorce or separation with a close friend?") related to the probability that the marriage might end in divorce. Possible responses ranged from 1 (*never*) to 4 (*yes, within the last 3 months*). The alpha coefficients for wives and husbands were .85 and .82, respectively.

Economic hardship—To estimate families' economic situation, an income-to-needs ratio was calculated using guidelines from the U.S. Department of Health and Human Services. This measure was computed by dividing total family income from all sources by the poverty

threshold based on household size and number of children under 18 (U.S. Bureau of the Census, 1990).

Results

Descriptive Statistics and Correlations

Analyses were conducted using the Statistical Package for Social Science, version 22 (SPSS Inc., Chicago, Ill., USA). Descriptive statistics and bivariate correlations among study variables are presented in Table 1. We first compared *SCL-90-R* depression and anxiety scores with the United States normative data reported by Derogatis (1983). Depression scores for mothers ($M = 1.58$, $SD = .49$) and fathers ($M = 1.45$, $SD = .42$) in our sample were higher than those of the normative adult sample ($M = 1.36$, $SD = .44$), $t(1,367) = 8.11$, $p < .001$ for mothers, $t(1,367) = 3.47$, $p < .001$ for fathers. At T1, adolescents in our sample had slightly lower depression scores ($M = 1.61$, $SD = .58$) than the normative adolescent sample ($M = 1.80$, $SD = .69$), $t(1,199) = 4.72$, $p < .001$. At T1, adolescents also had lower anxiety scores ($M = 1.49$, $SD = .51$) than the normative adolescent sample ($M = 1.66$, $SD = .62$), $t(1,199) = 4.72$, $p < .001$. At T2, young adult offspring in our sample were slightly more depressed ($M = 1.43$, $SD = .52$) than the normative adult sample ($M = 1.36$, $SD = .44$), $t(1,367) = 2.53$, $p = .012$, while anxiety scores ($M = 1.22$, $SD = .38$) were lower than the normative adult sample ($M = 1.30$, $SD = .37$), $t(1,367) = 3.60$, $p < .001$. Consistent with theoretical expectations, paternal depressive symptoms were significantly and positively correlated with youth depressive and anxiety symptoms in early adulthood, thus justifying the formal model testing that follows.

Predictors of Youth Internalizing Symptoms in Early Adulthood

Table 2 shows a series of hierarchical multiple regression analyses used to examine the effects of paternal depressive symptoms at T1 on youth depressive and anxiety symptom outcomes at T2. Analyses were run separately for each outcome. To determine whether paternal depressive symptoms had a unique influence on youth internalizing symptoms, variables were entered in two steps. Step 1 included adolescent gender, previous symptoms (type matched the dependent variable for each outcome), parental hostility, marital discord, economic hardship, and maternal depressive symptoms. Next, in the full model, paternal depressive symptoms were added; change in R^2 (ΔR^2) was used to assess the magnitude of association between paternal depressive symptoms and youth outcomes in early adulthood.

Results indicated good overall model fit for youth depression, $F(9, 385) = 5.11$, $p < .001$, and anxiety outcomes, $F(9, 385) = 2.65$, $p < .001$. Consistent with previous research on gender differences in the prevalence of depression (e.g., Van de Velde, Bracke, & Levecque, 2010), male offspring experienced fewer depressive symptoms than their female counterparts ($B = -.195$, $p < .001$). Internalizing symptoms in adolescence were a significant predictor of subsequent symptoms in young adulthood ($B = .149$, $p < .001$ for depression; $B = .092$, $p < .01$ for anxiety); these results are similar to previous longitudinal findings indicating high stability of mood disorders from adolescence into adulthood (Olino, Klein, Lewinsohn, Rohde, & Seeley, 2010; Roza, Hofstra, van der Ende, & Verhulst, 2003).

Consistent with results from Goodman et al. (2011) in their meta-analysis reviewing associations between maternal depression and children's adjustment, maternal depressive symptoms were a significant predictor of subsequent youth depressive symptoms (B = .094, p = .047). Maternal depressive symptoms were not significantly associated with offspring anxiety symptoms. Consistent with growing evidence indicating increased risk for internalizing psychopathology among children of depressed fathers (e.g., Bögels, & Phares, 2008; Field et al., 2010; Greene et al., 2008; Klein et al., 2005; Kane & Garber, 2009; Ramchandani & Psychogiou, 2009; Ramchandani et al., 2005, 2008; Reeb et al. 2010; Spector, 2006, Wanless, Rosenkoetter, & McClelland, 2008), results show that paternal depressive symptoms made significant contributions to children's depressive (H_1; B = .117, p = .030) and anxiety symptoms (H_2; B = .134, p = .002) in young adulthood, even after taking youth gender, previous symptoms, family functioning, economic hardship, and maternal depressive symptoms into account.

A moderated regression framework (Aiken & West, 1991) was used to examine whether the effects of paternal depressive symptoms on offspring internalizing symptoms varied as a function of maternal depressive symptoms or child gender. In our statistical models ($y_i = \beta_0 + \beta_1 X_{1i} + \beta_2 X_{2i} + \beta_3 X_{3i} + \varepsilon_i$), the outcome variable y_i (T2 youth outcome) was examined as a function of independent variables in the model (X_1 = paternal depressive symptoms; X_2 = maternal depressive symptoms or child gender), and the moderating effects indicated by an interaction ($X_3 = X_1 * X_2$). All continuous independent variables were grand mean centered. After product terms were created, a hierarchical multiple regression equation was constructed to test for moderator effects (Cohen, Cohen, West, & Aiken, 2003). Statistical controls and predictor variables were entered first. Next, product terms were added to the model and ΔR^2 was used to examine the significance of increment in explained variance (Baron & Kenny, 1986). Contrary to our expectations (H_3), maternal depressive symptoms did not moderate the effects of paternal depressive symptoms on offspring depressive (ΔR^2 = .001), t(384) = .52, *ns*, or anxiety symptoms (ΔR^2 = .003), t(384) = 1.11, *ns*. In addition, The Paternal Depression × Youth Gender interaction term (H_4) did not predict children's depressive (ΔR^2 = .000), t(384) = .05, *ns*, or anxiety symptoms (ΔR^2 = .003), t(384) = −.71, *ns*. For the sake of brevity, nonsignificant coefficients from moderated multiple regression analyses are omitted from the reported results.

Discussion

Fathers have been underrepresented in the literature on child psychopathology (Phares et al., 2005). However, a growing number of studies have documented increased risk for mental health problems among children of depressed fathers (for reviews, see Lefrancois, 2012; Ramchandani & Psychogiou, 2009; Spector, 2006). The majority of this research has used cross-sectional data and little attention has been given to the effects of paternal depression on youth outcomes beyond childhood. Using longitudinal data from a community sample, the central goal of this study was to examine the unique and prospective effects of paternal depressive symptoms on offspring internalizing symptoms in early adulthood. Consistent with conclusions drawn by Kane and Garber (2004) in their meta-analysis examining associations between paternal depression and children's psychopathology, we found that depressive symptoms in fathers predicted offspring risk for depressive and anxiety

symptoms. These associations were significant after controlling for effects of baseline youth symptoms, maternal depressive symptoms, and other potential confounding factors.

Research has only begun to examine the conditions under which paternal depression may have more or less of an effect on child outcomes. Contrary to previous findings indicating an interactive effect of maternal and paternal depression on child psychopathology (e.g., Brennan et al., 2002; Kahn et al., 2004), we found that associations between paternal depressive symptoms and youth outcomes did not differ as a function of maternal depressive symptoms. It is possible that the interactive effect of paternal and maternal depression may vary across different developmental stages; this is an important question for future research. Another proposed moderator of risk transmission is child gender (e.g., Goodman, 2007), although findings from previous research on gender-specific vulnerability have been mixed (Sheeber et al., 2002). In our analyses, associations between paternal depressive symptoms and youth outcomes in early adulthood were similar for boys and girls. Future longitudinal research is needed to identify the environmental factors and child characteristics associated with better or worse outcomes among offspring of depressed fathers.

The present study extends research on children of depressed fathers in several important ways. First, whereas the majority of existing data on children of depressed fathers have been cross-sectional, this study used a prospective, longitudinal design that included baseline youth internalizing symptoms as a statistical control. Therefore, our results reflect the predictive effect of paternal depressive symptoms on change in youth symptoms across an 8-year span from adolescence to early adulthood. Second, previous research on children of depressed fathers has focused primarily on risk transmission in infancy and childhood; we focused on offspring outcomes in early adulthood, which, for many individuals, is a high-risk period for the development of mood disorders (e.g., Olino et al., 2010). Third, not only did this study find that paternal depressive symptoms predicted youth internalizing symptoms above and beyond the effects of maternal depressive symptoms, but we also showed that associations were significant after controlling for other known correlates of father and child psychopathology, including parental hostility, marital discord, and economic hardship. A fourth strength of the current study was the use of multiple informants. In previous work, adult offspring have often been interviewed regarding the presence of psychopathology in their parents, thus presenting potential bias due to shared method variance.

Another contribution, as well as potential limitation, of the present study is the use of a community sample. Much of the previous research on children of depressed parents has relied on clinical samples, which may not be representative of the broader population of depressed individuals (Goodman et al., 1997). Moreover, evidence indicates that individuals with subdiagnostic depressive symptoms experience levels of psychological distress similar to those experienced by those with diagnosed depression (e.g., Sheeber, Davis, Leve, Hops, & Tildesley, 2007) and can benefit from clinical intervention (Allart-van Dam, Hosman, Hoogduin, & Schaap, 2003). However, community samples may not include many individuals experiencing diagnosable mood disorders; as such, further research on adult offspring of depressed fathers using data from clinical samples and other high-risk groups is warranted.

It is important to note several other limitations of the current study. First, results are limited to White, two-parent families. Future research will benefit from including more diverse populations, including single-parent, urban and minority families, and families with same-sex parents. A second limitation, as well as potential strength, is that our measure of parental hostility was based on observer ratings. Research has shown that children's perceptions of family relations often do not correlate with observer report (e.g., Cook & Goldstein, 1993) but are strongly related to their psychological adjustment (Forehand & Nousiainen, 1993; Plunkett, Henry, Robinson, Behnke, & Falcon, 2007; Sheeber et al., 2007). However, observational measures reduce the likelihood of shared method bias (see Richardson, Simmering, & Sturman, 2009). Third, our analyses did not consider changes in families' socioeconomic status over time, which have been associated with children's risk for developmental psychopathology (Martin et al., 2010; Miller & Taylor, 2012; Singh & Ghandour, 2012). For example, in our data, it is possible that changes in parents' employment status over the course of the study could account for additional variance in offspring internalizing symptoms in early adulthood. Despite this limitation, it is important to note that we included families' income-to-needs ratio, which has been shown to be a valid indicator of economic hardship experiences (Bauman, 2002; Beverly, 2001; Iceland & Bauman, 2007; Mayer & Jencks, 1989). Fourth, although our measures of young adult outcomes captured changes in anxiety and depressive symptoms over time, we did not include other potential consequences of paternal depression. For example, recent studies using younger samples have found increased risk for behavioral problems in children of depressed fathers (e.g., Gross et al., 2008; Marchand-Reilly, 2012).

A central challenge of future longitudinal research on children of depressed fathers will be identifying the complex, interactive mechanisms that underlie the transmission of risk from one generation to the next. Building on Goodman and Gotlib's (1999) model of intergenerational depression transmission, Connell and Goodman (2002) proposed several mechanisms by which paternal depression may be associated with children's increased risk for psychopathology. First, the genetic contribution to internalizing psychiatric disorders is high. For example, the heritability of depression is likely to be at least 31%–42% (Sullivan, Neale, & Kendler, 2000), while an estimated 30%–50% of the variability in risk for anxiety disorders is due to genetic factors (Gordon & Hen, 2004). Genotype variations could also influence the transmission of risk for internalizing psychopathology through predispositions to stress sensitivity (i.e., gene by environment interactions; see Rutter, 2003). Another potential mediator of risk transmission involves family functioning. Families with a depressed parent are less cohesive and express less care for each other than families with non-depressed parents (Kaslow, Deering, & Racusin, 1994), and a growing body of work has associated fathers' depression with child neglect (Lee, Taylor, & Bellamy, 2012), a lack of motivation to parent (Thomas & Kalucy, 2003), and hostile, intrusive, retaliatory, and coercive parenting behaviors (Wilson & Durbin, 2010). While research to date on interpersonal mechanisms of children's risk in relation to parental depression has focused almost exclusively on mothers, some cross-sectional evidence has been consistent with a mediational model of father–offspring conflict (Ge, Conger, Lorenz, Shanahan, & Elder, 1995; Kane & Garber, 2009). Taken together, theory and findings indicate that future research is warranted on complex models of family processes in order to develop a more

comprehensive understanding of the role fathers play in the psychological development of their children across the lifespan.

In conclusion, the present study highlights the importance of including fathers in research on developmental psychopathology, providing evidence that the unique and adverse impact of paternal depression on offspring mental health may extend well beyond the childhood years. Interventions aimed at preventing or reducing youth risk for internalizing problems during the transition to adulthood will benefit from future research on the additive and interactive genetic and environmental factors that mediate the intergenerational transmission of risk for psychopathology in children of depressed fathers. In addition, it is important to note that our data do not indicate inevitable offspring maladjustment as a consequence of paternal depression; further investigation of moderating factors is needed to understand the conditions under which many children of depressed fathers are able to adapt to their environment. Although the generalization of our findings to clinical populations is limited, these results suggest that treatments and preventive interventions designed to alleviate the antecedents and consequences of depression and anxiety disorders during the transition to adulthood may benefit from an approach that incorporates the needs of the family as a whole (LeFrancois, 2011; Reupert & Maybery, 2007). In this regard, clinicians may wish to consider the careful screening of psychiatric symptoms among adolescents and young adults whose fathers are depressed or have experienced depressive disorders in the past.

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Table 1

Descriptive Statistics and Intercorrelations Among Study Variables

Variable	*M*	*SD*	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*
1. Gender[a]	.46	.50	—	–.13*	–.01	–.03	–.01	–.10*	–.12*	.02	–.03	–.05	–.22**	–.09
2. T1 youth depressive symptoms	1.61	.58		—	.76*	.06	–.01	.19**	.13**	.04	–.15**	.08	.21**	.17**
3. T1 youth anxiety symptoms	1.49	.51			—	.08	–.04	.16	.14**	.04	–.15**	.08	.13*	.14**
4. T1 observed mother hostility	1.93	.91				—	.35**	.10*	.08	–.10*	.06	.05	.02	.02
5. T1 observed father hostility	2.06	.97					—	.07	.14**	–.09	.02	.06	–.05	–.04
6. T1 mother report marital discord	1.56	.63						—	.56**	.01	.33**	.03	.11*	.08
7. T1 father report marital discord	1.49	.56							–	–.02	.15**	.20**	.06	.05
8. T1 economic hardship	2.94	2.06								—	–.10	–.11*	–.04	–.04
9. T1 maternal depressive symptoms	1.58	.49									–	.09	.14**	.09
10. T1 paternal depressive symptoms	1.45	.42										—	.12*	.16**
11. T2 youth depressive symptoms	1.43	.52											—	.71**
12. T2 youth anxiety symptoms	1.22	.38												—

Note. (*N* = 395). T = Time. Psychiatric symptoms assessed using the *SCL-90-R* Depression and Anxiety Scales.

[a](0 = *female*, 1 = *male*).

*$p < .05$.

**$p < .01$.

Table 2

Hierarchical Linear Regression Analyses Predicting Youth SCL-90-R Internalizing Symptoms at Time 2

	Depression						Anxiety					
	Model 1			Model 2			Model 1			Model 2		
Predictors	***B***	***SE B***	**β**	***B***	***SE B***	**β**	***B***	***SE B***	**β**	***B***	***SE B***	**β**
Gender[a]	–.198	.051	–.188***	–.195	.051	–.186***	–.063	.039	–.082	–.060	.038	–.078
Previous youth symptoms	.155	.045	.171**	.149	.045	.165***	.098	.039	.130**	.092	.038	.122**
Observed mother hostility	.007	.030	.013	.006	.030	.011	.006	.023	.015	.005	.022	.011
Observed father hostility	–.036	.028	–.066	–.037	.028	–.068	–.025	.021	–.064	–.026	.021	–.066
Mother report marital discord	.030	.052	.036	.042	.052	.051	.021	.039	.034	.034	.039	.056
Father report marital discord	–.012	.055	–.013	–.034	.056	–.037	.004	.041	.006	–.021	.042	–.031
Economic hardship	–.010	.012	–.041	–.008	.012	–.032	–.009	.009	–.046	–.006	.009	–.033
Maternal depressive symptoms	.102	.056	.095*	.094	.056	.087*	.038	.042	.048	.028	.042	.036
Paternal depressive symptoms	—	—	—	.117	.062	.094*	—	—	—	.134	.047	.148**
R^2		.098***			.107***			.038*			.058**	
ΔR^2					.008*						.020**	

Note. (*N* = 395). Predictors were measured at Time 1.

[a](0 = *female*, 1 = *male*).

*$p < .05$

**$p < .01$

***$p < .001$ (1-tailed tests).