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# Transactions between maternal and child depressive symptoms emerge early in life

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## Abstract

**Objective:** Maternal depression is a robust risk factor for children's internalizing symptoms, however the intergenerational transmission of mood disorders is likely more complex than unidirectional, parent-directed effects. Theoretical models support transactional associations between maternal and child symptomatology over time, but have not been well examined, especially in younger, high-risk samples. The present investigation examined predictive transactional relations between maternal depression and children's internalizing in toddlerhood and early childhood using a cross-lagged panel model.

**Method:** Participants were 162 low-income, largely racial/ethnic minority mothers and their offspring (32% African American, 16% White, 52% Multiethnic/Other; 53% female) who were assessed when children were 18 months and 4 years old.

**Results:** There were significant cross-sectional relations between maternal depressive and child internalizing symptoms when children were 18 months, but not 4 years of age. Cross-lagged associations were evident such that maternal depression symptoms at 18 months were positively associated with internalizing symptoms among children at 4 years, adjusting for prior maternal symptom levels and the cross-sectional correlations between maternal-child symptoms at 18 months. Within the same model, children's internalizing symptoms at 18 months were also positively associated with maternal depressive symptoms at 4 years, adjusting for prior child symptom levels and cross-sectional correlations.

**Conclusions:** This study is among the first to demonstrate that transactional relations between maternal and child mood symptoms occur as early as toddlerhood/early childhood. Findings highlight the potential utility of inclusive, family-focused interventions that support both parents and children in the treatment of early emotional problems.

Internalizing disorders, which include depressive and anxiety disorders, are among the most pervasive forms of childhood psychopathology in community samples (Kovacs & Devlin, 1998). Despite early-onset and childhood prevalence rates that rival those observed later in

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life, research of internalizing disorders typically focuses more on adolescence than earlier periods of development (Ashford, Smit, van Lier, Cuijpers, & Koot, 2008). Young children were once thought to lack the maturity to experience internalizing disorders and if present at early ages, such symptoms were often deemed non-specific or transient (Luby, 2013). Such assumptions have largely been refuted through the development of improved assessment tools and completion of representative epidemiological studies (Luby, Belden, Pautsch, Si, & Spitznagel, 2009; Luby, Si, Belden, Tandon, & Spitznagel, 2009). Research on early internalizing is particularly important given findings that childhood-onset symptoms predict greater impairment, a poorer and more protracted course, and increased risk for comorbid disorders across the lifespan compared to symptoms that emerge later in life (Briggs-Gowan & Carter, 2008; Fanti & Henrich, 2010; Mesman, Bongers, & Koot, 2001).

There are numerous pathways through which children may develop internalizing symptoms and research has robustly identified maternal depression as one of the most potent risk factors. Associations of mood between maternal-offspring dyads have been observed across a range of developmental periods, including greater negative affect and difficult temperament during infancy (Feldman et al., 2009; Field, 2010) and internalizing problems during childhood (Fihrer, McMahon, & Taylor, 2009; Lengua & Kovacs, 2005; van der Waerden et al., 2015) and adolescence (Murray et al., 2011). Moreover, the negative effects of maternal depressive symptoms emerge in the context of clinical diagnoses of depression as well as at subthreshold symptom levels (Cummings & Davies, 1999; Goodman et al., 2011). Following from the well-known primacy of the early maternal-child relationship and the compromising effects of maternal depressive symptoms on formative attachment bonds, it has been suggested that exposure to maternal symptomatology during early childhood may be particularly detrimental (Connell & Goodman, 2002; Goodman et al., 2011). Illustratively, longitudinal prospective studies have observed that maternal depression exposure during infancy and toddlerhood is associated with internalizing problems in adolescence (Bureau, Easterbrooks, & Lyons-Ruth, 2009; Murray et al., 2011), even after adjusting for the effects of mothers' symptomatology in later childhood (Bureau et al., 2009).

Although research examining the influence of maternal depression on early internalizing implies a unidirectional causal pathway from parent to child, such relations may be more complex. Children are neither passive recipients of parental behaviors nor do parents operate in isolation from the behavioral responses of their offspring. Rather, parental functioning influences children's behavior and variability in children's behavior, in turn, elicits different types of parental behavior. Variously termed reciprocal, bidirectional, or transactional, such contingencies describe how processes of mutual influences between parents and offspring contribute to outcomes within the family context (Paschall & Mastergeorge, 2016). The concept of bidirectionality between parents and children has a lengthy theoretical history. Over 40 years ago, Sameroff criticized unidirectional, parent-directed models for being erroneously one-sided and mistakenly placing fault on mothers for "causing" child maladjustment (Sameroff, 1975). As an alternative, Sameroff emphasized dynamic transactions between children and the familial environment in which they are embedded; neither could be considered as operating independently of the other and both were equally deterministic of developmental outcomes (Sameroff, 2000; Sameroff &

Mackenzie, 2003). Belsky's model of the determinants of parenting also bears relevance to studies of bidirectionality, as it was one of the first to suggest that child characteristics influence parental functioning, which in turn, affects early development (Belsky, 1984). Belsky focused primarily on the influence of child temperament on parenting, particularly "those behavioral styles that make parenting more or less difficult" (p. 86). Although not specific to relations between maternal and child mood disorders, his model provides a strong foundation for examining such relations in a transactional framework.

Despite decades of *theoretical* support, there has been a persistent scarcity in *empirical* research of bidirectional parent-child associations compared to studies of unidirectional parental influences on children's behavior (Paschall & Mastergeorge, 2016). This may be particularly true in the context of research on maternal and child mood disorders; the limited extant investigations of bidirectionality have generally focused on relations between *parenting* and children's *externalizing symptoms* (Hipwell et al., 2008). One of the most well-researched examples of such reciprocal effects between parenting and child behavior is based upon Patterson's coercive model (Patterson, 1982), which describes a process that begins with the influence of harsh parenting on children's oppositional behaviors. Parents' subsequent attention and/or acquiesce to offspring defiance serves as inadvertent reinforcement that heightens offspring use of aversive behavior, then increasing parental frustration and return to harsh practices. There is strong empirical support for reciprocal relations between harsh, coercive parenting and children's noncompliant behavior (Combs-Ronto, Olson, Lunkenheimer, & Sameroff, 2009; Pardini, Fite, & Burke, 2008; Patterson, 2002; Snyder, Cramer, Afrank, & Patterson, 2005).

When transactional models have been used to examine relations between parent and child mood disorders, samples have generally been comprised of older children and adolescents. Elgar and colleagues examined bidirectional relations in middle childhood, for example, and observed a stronger influence of children's internalizing on maternal depression than the reciprocal mother-to-child relation. Similarly, Jaffee and Poulton (2006) observed only child-directed effects on maternal depression at ages 5 and 7. Conversely, other research has found stronger effects of maternal depression relative to the reciprocal effects of child internalizing (Brown et al., 2017; Fanti, Panayiotou, & Fanti, 2013; Garber & Cole, 2010; Nicholson, Deboeck, Farris, Boker, & Borkowski, 2011; Nilsen, Gustavson, Roysamb, Kjeldsen, & Karevold, 2013). In the very few studies that have explored transactional models in earlier childhood, reciprocal associations between maternal depression and offspring internalizing symptoms were observed when children were between 2 and 3 years old, but relations were unidirectional over the next several years (mother-to-child only; Yan & Dix, 2014; child-to-mother only; Hails, Reuben, Shaw, Dishion, & Wilson, 2017). As children become increasingly exposed to other caregivers and social contexts with age (e.g., daycare, preschool, teachers), there may be an attenuation of the close coupling between maternal and offspring mood symptomatology. Meta-analyses of unidirectional models suggest that maternal depression may be more strongly associated with internalizing symptoms among female compared to male children (Goodman et al., 2011). However, little is known about the potential moderating effects of offspring sex on transactional relations, as it has more commonly been included as a covariate in bidirectional models of parent and child mood disorders.

Beyond helping to offset to the paucity of research on reciprocal relations during very early childhood and in the context of parental mental health, the current investigation is also notable for testing bidirectionality in an understudied, high risk population. One of the most prevalent of mental health disorders among adults in general (Compton, Conway, Stinson, & Grant, 2006; Ferrari et al., 2013), depression rates are higher among mothers and individuals of low-income, ethnic minority status (Chung, McCollum, Elo, Lee, & Culhane, 2004; Ertel, Rich-Edwards, & Koenen, 2011; Weissman et al., 2004). Few studies have specifically examined transactional models in minority, economically disadvantaged families even though risk factors for children's mental health problems may not operate in the same manner across varied sociodemographic contexts (Garcia Coll et al., 1996). While there is some evidence to suggest stronger relations between maternal depression and children's internalizing within ethnic minorities families and those experiencing financial hardship, such conclusions must be tempered by the predominance of Caucasian, middle-to-high income samples from which results were drawn (Goodman et al., 2011). In fact, other research has observed nonsignificant or negative relations between traditional familial risk factors and early internalizing symptoms among ethnic minorities (Anderson & Mayes, 2010; Vendlinski, Silk, Shaw, & Lane, 2006). Leveraging a unique dataset of predominantly ethnic minority, low socioeconomic status women who were recruited for a prenatal mindfulness intervention and a subsequent longitudinal study of their offspring, the present study tested a model of transactional relations between maternal depressive and children's internalizing symptoms from toddlerhood through preschool age. We hypothesized the presence of significant reciprocal relations such that: 1. Elevated maternal depressive symptoms at 18 months postpartum would be associated with elevated internalizing symptoms when children were 4 years of age and 2. Elevated internalizing symptoms among children at 18 months of age would be associated with elevated maternal depressive symptoms when children were 4 months of age. Potential differences in transactional relations on the basis of child sex were also investigated, however given scarce research in this area, we refrained from advancing specific hypotheses regarding the differences in the strength of associations between male and female offspring.

## Method

### Participants and Procedures

The present study drew participants from a larger, quasi-experimental, early proof-of concept study of the effects of an 8-week mindfulness-based stress reduction program designed to target psychological distress and prevent excessive gestational weight gain among low-income, overweight and obese pregnant women. Women with gestational age between 12 and 19 weeks who were able to attend eight weekly intervention sessions were eligible to enroll in the intervention group ($n$ = 89, 55%). Women with gestational age between 20 and 24 weeks or those who could not attend the weekly intervention sessions were eligible for treatment as usual ($n$ = 73, 45%). Thus, the groups did differ on gestational weeks at enrollment ($M$ = 14.8, $SD$ = 2.6 for the intervention group vs. $M$ = 20.0, $SD$ = 4.5 for treatment as usual, $p$ < .01), however there were no between-group differences in sociodemographic characteristics, including age, race/ethnicity, education, marital status, income, and parity. The parent project did not use randomized assignment because the

primary goal was to examine whether the intervention exerted a clinically significant signal on gestational weight gain prior to proceeding with a more demanding and costly randomized design (Epel et al., 2019). A subsequent study continued assessments of infants of women who participated in the original project to determine developmental trajectories of offspring physical and mental health. This project was reviewed by Institutional Review Board at the University of California, San Francisco. Mothers provided written informed consent prior to the collection of any data. Additional recruitment and study details are reported in Coleman-Phox et al., 2013.

Women were recruited into the study based on the following criteria: 1) English speaking, 2) 12 to 23 weeks pregnant, 3) Between the ages of 18 and 45, 4) Household income of less than 500% of the federal poverty level (per HHS guidelines in the Bay Area, this amounted to $91,550 for our median household size of 3; https://aspe.hhs.gov/2010-hhs-poverty-guidelines), and 5) Self-reported pre-pregnancy body mass index between 25 and 41 $kg/m^2$. Exclusion criteria included: 1) A medical condition that could influence gestational weight gain, 2) Current practice of mindfulness mediation more than once per week, 3) Multiple pregnancy, 4) Currently taking weight loss or diabetes-related drugs, antidepressants, antipsychotics, opiate drugs, or corticosteroids, or 5) History of gastric bypass surgery.

For complete details of participant flow, we refer the reader to Bush et al., 2017. There were 215 women enrolled in the original study, 94% (n = 202) of whom were eligible for the follow-up study of children's physical and mental health ((non-eligibility due to dropout (n = 5), miscarriage (n = 3), lost to follow-up (n = 3), fetal death (n = 1), and moved out of the area (n = 1)). Of those eligible, the follow-up study enrolled 162 mothers and infants (80%).

## Measures

**Demographics.—**Background characteristics (e.g., race/ethnicity, marital status, annual household income, education) were assessed via self-report during pregnancy. Household income was converted to the percent above poverty level in the United States in order to adjust for family size. Mothers in the final analytic sample were 28.03 years old (*SD* = 5.76) on average upon enrollment and self-identified as African-American (39%, *n* = 63), Latino (31%, n = 50), White (15%, *n* = 24), Other/Multiethnic (12%, *n* = 20), Asian (3%, *n* = 4), or Pacific Islander (<1%, *n* = 1). Approximately one-third had a high school education or less, half had vocational training or some college, and the remainder of women had a college or advanced degree. Income ranged from less than $10,000 to $99,000 per year. Approximately 75% of the sample earned less than $40,000 per year for a median household size of 3 individuals, which is particularly low given that sample was drawn from an urban area with one of the highest costs of living in the United States. Approximately 50% of the sample lived at less than 100% above the federal poverty line (*Range* 15–794%). The sex distribution of children was approximately equal (53% female) and most were full-term at birth (95% full-term, *M* gestational age = 39.6 weeks, *SD* = 9.9). See Table 1 for full demographic and descriptive statistics on key study variables.

**Maternal depressive symptoms.—**Depressive symptoms were evaluated with maternal self-report on the Patient Health Questionnaire (PHQ-9) that was administered when offspring were 18 months and 4 years old. The PHQ-9 is a well-established and widely used measure of depressive symptom severity in the previous two weeks that has been validated in low-income and ethnically diverse samples (Huang, Chung, Kroenke, Delucchi, & Spitzer, 2006; Sidebottom, Harrison, Godecker, & Kim, 2012). In the current sample, Cronbach's alpha was 0.85 at 18 months and 0.82 at 4 years. Depressive symptoms were analyzed as continuous variables in all models, however for descriptive purposes, we report the distribution of the sample across depressive symptom severity categories (Kroenke, Spitzer, & Williams, 2001). When children were 18 months old, 57% of women reported none/minimal depressive symptoms (a score of 0 to 4 on the PHQ-9), 27% reported mild symptoms (a score of 5 to 9 on the PHQ-9), 8% reported moderate symptoms (a score of 10 to 14 on the PHQ-9), 8% reported moderately severe symptoms (a score of 15 to 19 on the PHQ-9), and <1% reported severe symptoms (a score of 20 or above on the PHQ-9). When children were 4 years old, 62% reported none/minimal symptoms, 26% reported mild symptoms, and 11% reported moderate symptoms, 1% reported moderately severe symptoms, and <1% reported severe symptoms.

**Children's internalizing symptoms.—**When children were 18 months and 4 years old, mothers reported on internalizing symptoms using the Child Behavior Checklist (CBCL) for ages 1.5 to 5 years (Achenbach & Rescorla, 2000). The CBCL is one of the most widely-used measure for assessing children's behavior problems and similar to the PHQ-9, has also been validated for use in socioeconomically and ethnically diverse samples (Gross et al., 2006). The internalizing scale of the CBCL consists of 36 items that are rated on a 3-point scale (0 = Not true to 2 = Very true or often true). Cronbach's alpha on the internalizing subscale of the CBCL was 0.81 and 0.88 when children were 18 months and 4 years old, respectively. As with maternal depressive symptoms, children's internalizing symptoms were modeled continuously to capture associations across the continuum of symptoms, however we report the frequency of children with subclinical or higher levels of internalizing (greater than or equal to a standardized *T*-score of 60 on the CBCL) for descriptive purposes: 15% at 18 months of age and 8% at 4 years of age.

## Data Analysis

**Missing data.—**There was some attrition between the 18 month and 4 year assessments. Among both mothers and children, 70% had data at both timepoints. Missing data was handled using full information maximum likelihood (FIML), which enabled Mplus to make use of all available data. As is standard practice when using FIML in Mplus, participants were retained if missing data were present for the predictors/covariates, but were not retained if missing data were present for the dependent variables (issues with reliability may emerge when participants with missing outcome data are retained). Comparing mothers and children with complete data to mothers and children without complete data, there were no significant differences on demographic factors, including maternal age, marital status, income, race, treatment group, or child sex (all $p$'s > .10). There were also no differences in maternal depressive symptoms or child internalizing symptoms at 18 months

when comparing mothers and children with complete data to mothers and children with data at 18 months only (*p*'s ranging from .21 to .98).

**Primary analyses.—**Cross-lagged panel analysis was used to examine the transactional relations between maternal depressive symptoms and their offspring's internalizing symptoms when children were 18 months and 4 years old. Use of a cross-lagged panel model allows for simultaneous estimation of cross-sectional correlations between constructs measured at the same time (e.g., maternal depressive and child internalizing symptoms at 18 months), autoregressive paths within each construct over time (e.g., maternal depressive symptoms at 18 months to maternal depressive symptoms at 4 years), and cross-lagged paths between constructs over time (e.g., maternal depressive symptoms at 18 months to child internalizing symptom at 4 years and child internalizing symptoms at 18 months to maternal depressive symptoms at 4 years). Thus, the cross-lagged effects in the present model can be interpreted as the predictive influence of maternal depressive symptoms (or child internalizing) at 18 months on change in child internalizing (or maternal depression) at 4 years of age, controlling for prior levels of each construct and their previous cross-sectional correlation. The standardized beta values of the cross-lagged effects can be interpreted such that higher coefficients indicate greater relevance to the predictor. Model fit was evaluated using a) root mean squared error of the approximation (RMSEA) with 90% confidence intervals, b) comparative fit index (CFI), c) Tucker-Lewis index (TLI), and d) chi-square with degrees of freedom and *p* (Hu & Bentler, 1999). Maternal race/ethnicity, age, marital status, household poverty, child sex, and treatment group were included as covariates in our final models. All analyses were conducted in Mplus version 8.1.

To test for potential differences in the transactional relations of maternal depression to child internalizing between male and female offspring, we also estimated two multiple group models in which the path coefficients were freely estimated or constrained to be equal across the sexes. The two models were compared with the $\chi^2$ difference test, with a significant result ($p < .05$) indicating poorer model fit when paths were constrained to be equal and providing evidence for moderation by child sex.

## Results

Fit indices for the cross-lagged model presented in Figure 1 indicated a good fit of the model to the data (RMSEA = 0.00 (90% CI = 0.00, 0.13), CFI = 1.00, TLI = 1.07, $\chi^2(4) = 3.28$, $p = 0.51$). There was a significant association between maternal depressive and children's internalizing symptoms at 18 months ($\beta = 0.436$, $p < .01$), however symptoms were no longer correlated between dyads when offspring were 4 years old ($\beta = 0.047$, $p = .64$). Autoregressive paths from 18 months to 4 years indicated significant stability of both maternal depressive symptoms ($\beta = 0.411$, $p < .01$) and children's internalizing symptoms ($\beta = 0.292$, $p < .01$). Even after controlling for stability (cross-sectional associations between maternal and child symptoms and symptom levels among children at 18 months), maternal depressive symptoms at 18 months was associated with change in offspring internalizing symptom at age 4 ($\beta = 0.222$, $p < .01$). In other words, elevated levels maternal depressive symptoms at 18 months were associated with increased child internalizing several years later. Similarly, significant relations were also observed in

the reciprocal cross-lagged paths from children's internalizing at 18 months to maternal depressive symptoms at 4 years: higher levels children's internalizing predicted increases in later maternal depressive symptoms, adjusting for the stability of maternal depression and the correlation between maternal and child symptoms at 18 months ($\beta = 0.371$, $p = .016$). Comparing the coefficient values of the cross-lagged paths, there was a larger coefficient relating children's internalizing at 18 months to maternal depressive symptoms at 4 years ($\beta = 0.371$) compared to the coefficient relating maternal depressive symptoms at 18 months to later child internalizing ($\beta = 0.222$), though formal tests of the difference did not reach statistical significance; $\chi^2(1) = 2.214$, $p = .10$).

Lastly, a multiple group model tested the equivalency of the reciprocal, transactional relations across male and female offspring. The baseline multigroup model provided adequate fit to the data, $\chi^2(8) = 13.08$, $p = 0.10$; $\chi^2/df = 1.64$; CFI = 0.95; RMSEA = 0.106 (90% confidence interval = 0.000–0.205). Equality constraints were specified to test for gender differences in the main model (not on relationships with covariates). An equality constraint on the path from maternal depressive to children's internalizing symptoms did not improve model fit, $\Delta\chi^2(6) = 10.35$, $p = 0.116$, indicating the path was not moderated by sex.

## Discussion

The present study tested a longitudinal, transactional model of relations between mothers' depressive symptoms and children's internalizing symptoms measured when children were 18 months and 4 years of age. Findings suggest maternal and child mood symptoms are reciprocally related during the early years of life. Heightened levels of maternal depressive symptoms in late infancy/early toddlerhood were associated with increases in preschool-age children's internalizing and in a parallel fashion, heightened internalizing symptoms among offspring in late infancy were related to increases in maternal depressive symptom when their children were 4 years old. By using cross-lagged panel analysis, we are able to draw stronger inferences regarding temporal associations between maternal depressive symptoms and changes in children's internalizing (and vice versa) that cannot be attributed to within-individual symptom stability and the prior association between maternal-child symptoms.

Results of the present study provide support for theoretical models of transactional associations between parents and offspring and extend such frameworks in several important ways. First, extant empirical studies have largely applied longitudinal, reciprocal models to understanding associations between negative parenting practices and children's externalizing symptoms. Despite the sensible nature and intuitive appeal of transactional relations between symptoms of maternal depression and children's internalizing, prior studies have mainly explored unidirectional, mother-to-child depression effects (Paschall & Mastergeorge, 2016). Even studies of maternal depressive symptom chronicity and/or timing of exposure have not considered how such longitudinal relations may be influenced by the reciprocal effects of children's own internalizing symptoms (Brennan et al., 2000; Hammen & Brennan, 2003; Petterson & Albers, 2001). The few studies that have examined bidirectional relations between maternal-offspring mood symptoms have mostly employed samples of older children and adolescents (e.g., Kouros & Garber, 2010; Hughes & Gallone, 2010). To

our knowledge, the present study is among the first to document transactional associations beginning within the first couple years of life.

Research on the temporal associations between maternal and offspring mental health within ethnic minority families is also scarce (Bagner, Pettit, Lewinsohn, Seeley, & Jaccard, 2013). The present study provides important documentation about the nature of such associations in a sample that is at heighted risk for poor outcome and disparities in mental health treatment due to ethnic minority and low socioeconomic status (Alegría et al., 2008; Petterson & Albers, 2001; Santiago, Kaltman, & Miranda, 2013). Use of such underrepresented samples has been deemed particularly important in the context of research of maternal depression and offspring adjustment because it "bridges" studies focused on either clinical or community populations and provides a different perspective in terms of risk and prevalence rates (Burt et al., 2005). Although the prevalence of moderate to severe depressive symptoms among mothers in the present sample was generally consistent, if not slightly elevated, as compared to the general population (approximately 12–16% versus 9%; Martin, Rief, Klaiberg, & Braehler, 2006; Strine et al., 2008; Vilagut, Forero, Barbaglia, & Alonso, 2016), rates of children's elevated internalizing symptoms in the current sample were somewhat low, particularly when children were 4 years old (15% when children were 18 months old, 8% at 4 years old). Using comparable checklist measures, prior studies of community samples of toddlers and preschool-age children reported rates of internalizing ranging from 6–17% (Ashford et al., 2008; Beyer, Postert, Müller, & Furniss, 2012; Briggs-Gowan, Carter, Bosson-Heenan, Guyer, & Horwitz, 2006; Lavigne et al., 1996). Given sociodemographic risk factors in the current study, we may expect higher rates of children's internalizing symptoms. Although it is possible that the lower-than-expected rates of early internalizing are due to maternal underreporting, it is notable that rates of prematurity in this sample were also much lower than current national averages (5% in the current sample versus the national average of 10%; Hamilton, Martin, Osterman, Driscoll, & Rossen, 2018). Thus, there may protective or resilience-promoting factors that are operating to support children's healthy development across various domains. Nonetheless, the lower rates of internalizing do not preclude the significant transactional effects observed in the present study.

Much has been written about the mechanisms through which maternal depression influences children's risk for adverse outcomes, including internalizing symptoms. For example, depression-induced deficits in positive parenting and the quality of the mother-child relationship have been consistently implicated in the causal pathways leading to children's internalizing (Goodman et al., 2011; Herba, Glover, Ramchandani, & Rondon, 2016; Lim, Wood, Miller, & Simmens, 2011). Other research has suggested that offspring exposed to maternal depression are at risk for mental health problems due to negative effects on children's emotion regulation strategies (Suveg, Shaffer, Morelen, & Thomassin, 2011), disruptions to the interparental relationship (Lim et al., 2011), and low levels of maternal support (McCarty & McMahon, 2003). Given that the first several years of life are a sensitive period for the development of behavioral self-regulatory capacities (Bush & Boyce, 2016; Kochanska, Coy, & Murray, 2001) and learning/parental modeling is high (Fisak & Grills-Taquechel, 2007), disruptions in the maternal-offspring relationship may influence the onset and maintenance of children's mood disorders. Finally, maternal depressive symptoms

during the early childhood years may reflect, at least in part, enduring symptomatology from the prenatal period (Heron et al., 2004). As maternal depression during pregnancy has been associated with dysregulation of offspring neurobiological functioning in ways that may elevate the risk for early mental health problems, these reciprocal relations may have fetal origins (Bilbo & Schwartz, 2012; Glover, O'Connor, & O'Donnell, 2010; Martini, Knappe, Beesdo-Baum, Lieb, & Wittchen, 2010).

The pathways that link children's internalizing symptoms to increased levels of maternal depression have received less attention and may be somewhat less obvious. Behavioral correlates of children's internalizing (e.g., withdrawal, lack of communication, limited engagement in social interactions) may compromise positive qualities of the maternal-child relationship and be experienced by mothers as stressful, contributing to her depressive symptoms (Dix & Yan, 2014). Illustratively, associations between children's withdrawal behavior and maternal depressive symptom has been observed during toddlerhood (Yan & Dix, 2014). The presence of children's internalizing symptoms may also contribute to parent perceptions of low parenting self-efficacy. Mothers may attribute their child's behavior problems to deficits in their own functioning within the parental role, leading to lower parenting self-efficacy which in turn, has been associated with maternal depressive symptoms (Howell, Mora, DiBonaventura, & Leventhal, 2009). Finally, the negative consequences of childhood mental health symptoms may emerge at other levels of the socioecological system (e.g., associations with children's poorer peer relationships, isolation and lack of parental support networks, deficits in children's school functioning and subsequent difficult parent-teacher interactions). Strains across multiple childhood and familial environments may lead to chronically high levels of maternal stress and the development of depressive symptoms (Bouma, Ormel, Verhulst, & Oldehinkel, 2008). In sum, these maternal-to-child (and vice versa) relations can operate through varied pathways. The present study suggests that a transactional framework provides insights into reciprocal relations that support the maintenance and/or exacerbation of maternal and offspring mood symptoms over time.

Results of the present study must be considered in the context of several limitations. First, we relied on maternal report of both her own depressive symptoms and her child's internalizing symptoms. Though symptom reporting may be biased when only a single source of data is considered and by the presence of mental health symptoms, existing research suggests any biases in reporting of child symptomatology due to maternal depression are minimal (De Los Reyes & Kazdin, 2005). In addition, our analytic approach addressed such biases by controlling for prior maternal symptomatology. Second, the predominantly ethnic minority and socioeconomically disadvantaged nature of our sample marks an important contribution to extant literature that has mostly focused on Caucasian, middle-class families. However, such features may limit generalizability and the ability to test more nuanced models. In particular, the transactional nature of mother and child mood symptoms does not operate in a vacuum and qualities of the broader social environment may influence the manner in which intergenerational effects emerge. Samples that include a broader range of contextual factors are well-positioned to test the potential moderating role of socioeconomic status and other qualities of the environment on reciprocal relations. Additionally, we did not have a clinical sample or a diagnostic measures of children's

internalizing disorder or maternal depression, which may limit the applicability of findings to maternal-child dyads with more severe levels of psychopathology. Third, the present study was limited to two timepoints during early childhood (18 months and 4 years of age) and reciprocal relations may develop earlier in development. Testing such extended models is an important direction for future research. Finally, we focused primarily on maternal depressive symptoms in the current study, rather than parental symptoms more broadly. Prior studies have documented prevalence rates of depressive symptoms among fathers of young children that rival those observed among mothers (Paulson & Bazemore, 2010), including within other ethnic minority, high-risk samples (Roubinov, Luecken, Crnic, & Gonzales, 2014). As father involvement may increase from infancy through toddlerhood and early childhood (Lamb & Lewis, 2010; Yeung, Sandberg, Davis-Kean, & Hofferth, 2001) and recent research documents a trend of increasing paternal time in childcare activities (Hofferth & Lee, 2015), an understanding of transactional relations between paternal mental health and early child adjustment is an important gap to be explored in subsequent studies.

In conclusion, this study demonstrated reciprocal, transactional relations between maternal depressive symptoms and children's internalizing symptoms when children were 18 months and 4 years of age. These findings extend existing research of bidirectionality to a sensitive period of early development and to a high-risk, predominantly ethnic minority and economically disadvantaged sample. Perhaps most novel is the demonstrated link from children's early internalizing symptoms to later maternal depressive symptoms. These findings underscore the importance of repeated screening of *both* maternal and child mental health, particularly when one individual within the dyad is exhibiting heightened symptomatology. The American Academy of Pediatrics recently advocated for the screening and management of perinatal parental depression in *pediatric* healthcare settings (Rafferty et al., 2019) and results of the present study indicate the importance of extending such practices beyond the first postpartum year. Although prevention and intervention programs that focus on maternal depression or child internalizing may have cascading effects that lead to enhanced well-being among both members of the dyad, the reciprocal nature of such relations calls for integrative service models that attend to both maternal and child mental health. However, such approaches remain less common than therapies that focus on the individual child or parent (Zalewski, Goodman, Cole, & McLaughlin, 2017). Even when treatments for child mental health issues involve parents, it is often done from the perspective of improving parenting with little focus on parental mental health. A lack of concurrent attention to parental psychopathology may undermine the potential benefits of parenting interventions to improve child psychopathology (Zalewski et al., 2017). Optimal screening and intervention may benefit from a more family-centered approach that addresses the needs of the individual as well as those of the parent-child dyad.

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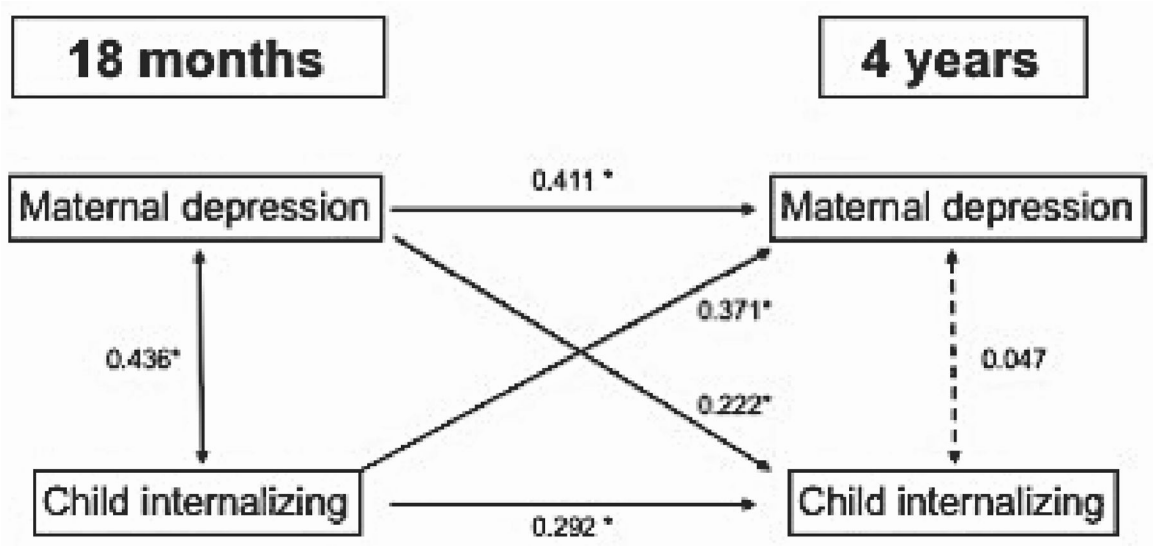


**Figure 1.**

Transactional model of the relations between maternal depressive and children's internalizing symptoms

Notes. Standardized coefficients shown. Solid lines denote significant paths and dashed lined denote nonsignificant paths.

**Table 1.**

Sample demographics descriptive statistics for key study variables (n = 162)

| | n (%) |
|---|---|
| Child sex | |
| Boys | 77 (47.5) |
| Girls | 85 (52.5) |
| Mother age (*M, SD)* | 28.03 (5.76) |
| Mother race/ethnicity | |
| African American | 63 (39) |
| Latina | 50 (31) |
| White | 24 (15) |
| Other/Multiethnic | 20 (12) |
| Asian | 4 (3) |
| Pacific Islander | 1 (<1) |
| Mother marital status | |
| Single | 52 (32) |
| Partnered or married | 109 (67) |
| Missing | 1 (<1) |
| Family Income | |
| Less than $10,000 | 41 (25) |
| $10,000–$19,999 | 38 (23) |
| $20,000–$29,000 | 29 (18) |
| $30,000–$39,000 | 17 (10) |
| $40,000–$49,999 | 8 (5) |
| $50,000–$59,999 | 7 (4) |
| $60,000–$69,999 | 5 (3) |
| $70,000–$79,999 | 7 (4) |
| $80,000–$89,999 | 2 (1) |
| $90,000–$99,999 | 1 (<1) |
| Missing | 7 (4) |
| Mother Education | |
| Less than high school | 16 (10) |
| High school diploma | 34 (21) |
| Vocational school | 9 (6) |
| Some college or 2-year degree | 72 (44) |
| College graduate | 25 (15) |
| Professional or graduate degree | 6 (4) |
| Maternal depressive symptoms at 18 months (*M, SD*) | 5.23 (4.91) |
| Maternal depressive symptom at 4 years (*M, SD*) | 6.61 (6.19) |
| Child internalizing symptoms at 18 months (*M, SD*) | 51.2 (9.0) |
| Child internalizing symptoms at 4 years (*M, SD*) | 46.2 (10.5) |

**Table 2.**

Zero-order correlations for key study variables

| Variable | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|
| 1. Maternal depressive symptoms (18 months) | .46** | .53** | .40** | −.01 | −.21* | .18 | −.08 | .04 | −.05 |
| 2. Child internalizing symptoms (18 months) | -- | .42** | .45** | −.25** | −.25** | .19* | −.13 | .19 | −.11 |
| 3. Maternal depressive symptoms (4 years) | | -- | .25** | −.14 | −.25** | .12 | −.15 | .03 | −.09 |
| 4. Child internalizing symptoms (4 years) | | | -- | −.06 | −.05 | −.08 | −.06 | −.01 | .08 |
| 5. Maternal race/ethnicity | | | | -- | .28** | −.01 | .12 | .10 | .47** |
| 6. Maternal age | | | | | -- | −.06 | .01 | −.11 | .28** |
| 7. Child sex | | | | | | -- | −.12 | −.03 | −.12 |
| 8. Treatment group | | | | | | | -- | −.02 | .07 |
| 9. Marital status | | | | | | | | -- | .29** |
| 10. Household poverty (percent above the poverty line) | | | | | | | | | -- |

*Note*. Race/ethnicity coded as 0 = non-White, 1 = White; child sex coded as 0 = boy, 1 = girl; treatment group coded as 0 = treatment as usual, 1 = mindfulness intervention; Marital status coded as 0 = single, separated, or divorced, 1 = married or in a relationship

* $p < .05$

** $p < .01$