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Testing the Temporal Relationship Between Maternal and Adolescent Depressive and Anxiety Symptoms in a Community Sample

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Abstract

Objective—Transactional models have been used to explain the relationship between maternal depression and child behavioral problems; however, few studies have examined transactional models for maternal depression and adolescent depression and anxiety.

Method—Using an autoregressive cross-lagged analysis, we examined the longitudinal association between maternal and adolescent depression to determine the extent to which maternal depression influences adolescent depression and anxiety, and vice versa, over the course of a four-year period. Participants were a community sample of 277 mother-adolescent dyads with offspring aged 10–14 at the first year used in the analyses (43.7% female; 35% African American, 2.9% Hispanic/Latino). Depressive symptoms were assessed using maternal self-report (Center for Epidemiological Studies-Depression Scale [CESD]; Radloff, 1977), and adolescent depression and anxiety were assessed by self-report (Revised Child Anxiety and Depression Scale [RCADS]; Chorpita, Yim, Moffitt, Umemoto, & Francis, 2000).

Results—The final model, $\chi^2(14) = 23.74, p = .05$; TLI = .97; CFI = .98; RMSEA = .05, indicated that maternal depression was significantly associated with adolescent depression two years later. Interestingly, adolescent depression did not significantly predict maternal depression, and the association between maternal and adolescent depression was not moderated by gender, age, or ethnicity. The association between maternal depression and adolescent anxiety was weaker than that observed for adolescent depression.

Conclusions—Results suggest that the transaction model of maternal depression may not extend to adolescent depression and anxiety. Furthermore, maternal depression can have an enduring effect on adolescent depression and continued research and clinical monitoring over extended periods of time is warranted.

Keywords

Depression; Parent-Child Relationships; Internalizing Disorders

Depression is a highly prevalent psychiatric disorder, with lifetime prevalence estimates ranging from 15% to 41% (Kessler, et al., 2005; Moffitt, et al., 2010); and depressive symptoms being reported in 20%–28% of mothers (Shaw, Connell, Dishion, Wilson, & Gardner, 2009). A wealth of data has demonstrated associations between maternal depression and early onset psychiatric and behavioral problems in their offspring (e.g., Billings & Moos, 1983; Goodman & Gotlib, 1999; Jaffee, et al., 2002; Trapolini, McMahon, & Ungerer, 2007; Weissman, Gammon, et al., 1987; Weissman, Warner, Wickramaratne, Moreau, & Olfson, 1987). Cross-sectional studies have found that, compared with offspring of non-depressed mothers, offspring of depressed mothers demonstrate greater physiological responses to stress (Lovejoy, Graczyk, O'Hare, & Neuman, 2000), and are at a greatly increased risk of developing a psychiatric disorder, particularly anxiety disorders, major depression, and alcohol dependence (e.g., Billings & Moos, 1983; Weissman, Gammon, et al., 1987). There is also evidence that offspring of depressed parents (including mothers and fathers) continue to experience serious psychological and behavioral problems even after the parent's depression has remitted (Billings & Moos, 1985).

Parental depression is thought to confer risk for offspring depression via multiple potential pathway including family environment/social learning mechanisms, cognitive processing mechanisms, and biological/genetic mechanisms (see Joormann, Eugene, & Gotlib, 2008 for a review). For example, research on family environment/social learning mechanisms has found that offspring of depressed mothers are often exposed to higher levels of family stress such as marital discord, ineffective or aversive behavior management, or modeling of poor emotion regulation (Abela, Zinck, Kryger, Zilber, & Hankin, 2009; Hammen, Brennan, & Shih, 2004; Hammen, Shih, & Brennan, 2004; Trapolini, et al., 2007). Studies have also found significant differences in cognitive processing mechanisms, such that offspring of depressed mothers tend to engage in biased information processing, negative attribution styles, and rumination more than offspring of non-depressed mothers (Alloy, et al., 2004; Flancbaum, et al., 2011; Gibb, Alloy, Abramson, Beevers, & Miller, 2004; Joormann, Talbot, & Gotlib, 2007). Biological and genetic mechanisms also play a significant role in risk for offspring depression. Depression has a genetic heritability from mother to offspring which may also convey risk for structural and functional differences in brain development and activity, and altered stress-reactivity and neuroendocrine systems (Gotlib, et al., 2010; Gotlib, Joormann, Minor, & Hallmayer, 2008; Joormann, Cooney, Henry, & Gotlib, 2012). Taken together, the three mechanisms likely interact with one another such that familial influence on child depression may be due to a complex combination of inherited vulnerabilities and environmental influence (e.g., stressful family environment, harsh parenting, etc., associated with parental depression).

Although the preponderance of the literature on the intergenerational transmission of depression has focused on the transmission *from* parents *to* offspring, transactional models have suggested that child behavior can influence maternal psychopathology. The transactional models of parent-child relationships in the development of psychopathology was first proposed by Sameroff and Chandler (1975). Since then, studies testing the reciprocal relationships of the transactional model have focused primarily on the effects of child *behavior* problems (Sameroff, 2009), such as oppositional defiant disorder on parenting behavior (Curran, Stice, & Chassin, 1997) or parental depression (Feng, et al., 2009; Keenan, Feng, Hipwell, & Klostermann, 2009). These studies, however, have not examined the temporal relationship between maternal and adolescent depression; this is a critical limitation to the extant literature. Given that internalizing and social withdrawal symptoms characterize depression, it is possible that offspring depression places fewer emotional and behavioral management demands on depressed mothers compared to youth with externalizing behavior problems. Understanding the temporal and directional relationship between maternal and youth *depression* represents an important, yet understudied, area of study.

Unfortunately, many of the studies examining the relationship between maternal and offspring depression are also confounded by the use of parent-report of youth symptoms. Research has found that depressed parents are more likely to report elevated youth behavioral and emotional problems compared to youth- and teacher-reports (Briggs-Gowan, Carter, & Schwab-Stone, 1996; De Los Reyes, Goodman, Kliewer, & Reid-Quinones, 2008; De Los Reyes & Kazdin, 2005). For example, a prospective study that examined the effects of maternal depression on children assessed at 4 months, 12 months, 15 months, and 4 years found a relationship between maternal depression and child internalizing and externalizing problems according to maternal report, but these relationships dropped from significance when teacher reports were used (Trapolini, et al., 2007). A recent meta-analysis found that the effect size between child psychopathology and maternal depression is significantly larger for maternal-report compared to teacher-report or child self-report of child psychopathology symptoms (Joormann, et al., 2007). Thus, associations with maternal depression may be artificially inflated by the depressed mother's perception of her child's behavior.

Compared to cross-sectional studies examining parent-child depressive symptoms, longitudinal studies are fewer in number. The extant longitudinal studies have suggested that offspring of depressed parents remain at increased risk for negative consequences across the lifespan (Goodman & Gotlib, 1999; Trapolini, et al., 2007; Weissman, et al., 2006). In a 20-year follow-up study, Weissman and colleagues (2006) determined that the offspring of depressed parents demonstrated significantly greater rates of depression, anxiety, substance use disorders, and physical health problems as adults. Although research has shown that child depression often occurs in close proximity to mother depression (Hammen, 1991), few have examined fluctuations in depression across time within mother-child dyads. One study by Oppenheimer and colleagues (2013) that did examine fluctuations in maternal depression across time in relation to child depression found that a variant of the serotonin transporter gene (5-HTTLPR) interacted with elevations in maternal depression to predict offspring depression. However, Oppenheimer and colleagues (2013) did not examine the transactional

effect of youth depression on maternal depression. Longitudinal data analytic strategies such as autoregressive models have the ability to examine individual changes in depression across time and can examine the extent to which maternal and child depression mutually influence one another. In one such study, Bagner and colleagues' (Bagner, Pettit, Lewinsohn, Seeley, & Jaccard, 2012) prospective study of depressed mothers and children demonstrated that parental depressive symptoms in a specified year predicted child behavior problems at the subsequent year; moreover, child behavioral problems also predicted parental depression, supporting the transactional model. However, analyses relied on parent report of child symptoms, which could have inflated the relationship between parent and child depression.

Further, Bagner and colleagues' (2012) work focused on children between the ages of four and seven, and it is unknown whether a similar longitudinal trajectory or transactional model of depressive symptoms exists among older children and adolescents, a critical developmental period in which rates of depression are higher compared to early childhood. Indeed, adolescence represents a particularly at-risk stage for depression, as incidence of the disorder tends to peak between the ages of 15–20 (Weissman, Warner, et al., 1987). Developmental and social changes during adolescence may alter the nature of the relationship between maternal and offspring depression. During this time, adolescents tend to spend less time with parents and more time with peers (Larson & Richards, 1991; Larson, Richards, Moneta, Holmbeck, & Duckett, 1996), and it is tempting to assume that parents may exert less influence over their child's behavior during this time. However, researchers have suggested that parents continue to exert an indirect influence on adolescents in terms of peer group affiliation and psychological and behavioral outcomes (Brown, Mounts, Lamborn, & Steinberg, 1993). Research has demonstrated that maternal depression during infancy and early childhood is associated with poorer mother-child attachment (Martins & Gaffan, 2000) and behavioral and psychological dysfunction into adolescence (Halligan, Murray, Martins, & Cooper, 2007). However, it is unknown the extent to which the relationship between depression in mothers and adolescents represents the continued effects of previous exposure to maternal depression, or if it represents an ongoing transaction between mothers and offspring (Joormann, et al., 2008). Examining the temporal course of maternal-offspring depression during adolescence is critical to untangle this question and to inform depression prevention and treatment efforts.

Current Study

The present study aimed to examine the association between maternal and adolescent depression over time in a community sample of mothers and adolescents using an autoregressive framework. This study addresses several gaps in the literature by 1) examining the association between maternal and adolescent depressive symptoms during the critical developmental period of adolescence, 2) utilizing autoregressive models to examine the temporal associations between these variables assessed yearly over a 4-year period, and 3) using self-report (as opposed to parent-report) of adolescent depressive symptoms. Based on previous research on the transactional model of maternal depression and child behavior problems, we hypothesized that: (1) concurrent associations would be observed between maternal and adolescent depressive symptoms, (2) that maternal depressive symptoms would predict subsequent adolescent depressive symptoms, and (3) that youth depressive

symptoms would predict subsequent maternal depressive symptoms. However, based on the use of self-report measures, we hypothesize that the magnitude of the relationship between maternal and adolescent depressive symptoms would be moderately attenuated compared to studies that have used only maternal report. We also included an exploratory analysis to test the specificity of effects by examining the extent to which maternal depression is associated with adolescent anxiety. Previous research has found that offspring of depressed mothers are more likely to report broad internalizing (e.g., Bagner et al., 2012; Trapolini et al., 2007) and anxiety symptoms (Halligan et al., 2007; Hammen & Brennan, 2003; Weissman et al., 2006), however, the longitudinal associations between maternal depression and offspring anxiety is unknown.

Given previous associations between demographic characteristics in the depression literature we examined gender, race, and income as covariates in the autoregressive model. Findings on the moderating effects of gender on the relationship between maternal and offspring depression have been inconsistent with regard to the influence of maternal depression exposure on boys, with some studies finding no relationship (Davies & Windle, 1997), and others finding that boys exposed to maternal depression are more likely to report internalizing symptoms as well as externalizing symptoms if also exposed to marital conflict (Essex, Klein, Cho, & Kraemer, 2003). Studies examining racial/ethnic differences in depression transmission have found some support for partial mediation of race/ethnicity in the relationship between maternal depression and offspring psychosocial development, leading to some speculation that cultural differences in parenting practices may account for this difference (Pachter, Auinger, Palmer, & Weitzman, 2006).

Methods

Subjects

Participants included mother and adolescent dyads ($n = 277$) from an ongoing prospective study of risk-taking behaviors in a community sample residing in the greater metropolitan Washington, D.C. catchment area recruited through media advertisements and mailings to local schools, libraries, and Boys and Girls Clubs (see MacPherson, Magidson, Reynolds, Kahler, & Lejuez, 2010; MacPherson, Reynolds, et al., 2010). Families were assessed yearly over the course of the study. As mother depression was added at the second wave of data collection, only data from Wave 2 (T1) through Wave 5 (T4) were used. Youth were aged 10 to 14 at T1. The University of Maryland Institutional Review Board reviewed and approved data collection. Child participants were 43.7% female ($n = 121$). Because there were not enough participants in each ethnic group to examine the effects of each ethnic group separately (35.4% African American, 2.9% Hispanic/Latino, 0.4% Native American, 1.4% Asian, 10.5% “Other”, 0.4% missing), ethnicity was dichotomized into “European-American” and “Non-European-American” (EA, non-EA). Of those reporting ethnicity, 49.3% ($n = 136$) were identified by their mother as EA, and 50.7% were identified as non-EA. The median family income at T1 was \$90,000 ($M = \$96,191$; $SD = \$54,440$), and was binned into quartiles for the analyses (\$0–48,000; 48,001–85,000; 85,001–120,000; 120,001–highest). According to the 2005–2009 American Communities Survey of the US Census Bureau (nearest report to T1 data collection), the median household income was

\$84,424 (M=\$108,302) for the Washington-Arlington-Alexandria, DC-VA-MD-WV metropolitan statistical area, and 51.2% were non-Hispanic White, 26.1% Black or African American, 8.4% Asian, 12.2% Hispanic or Latino, 2.5% two or more races. Thus, this sample was representative of the greater Washington DC area with regard to household income and percentage of EA participants. Within non-EA participants, African American participants were slightly over-represented, while Asian, Hispanic/Latino, and Native American participants were under-represented.

Measures

Revised Child Anxiety and Depression Scale (RCADS; Chorpita, et al., 2000)—

The RCADS is a 47-item youth self-report measure that assesses symptoms of several DSM-IV-TR anxiety and depressive disorders. It includes five anxiety subscales, a total anxiety scale (comprised of all anxiety subscale items), and major depressive disorder (MDD) scale. The 10-item MDD scale and the 37-item total anxiety (ANX) were used in the current analyses. Youth respond to questions (e.g., “I feel sad or empty”) using a 4-point scale: 0=never, 1=sometimes, 2=often, and 3=always, and scores were summed for analyses with higher scores representing greater depressive symptoms. Several psychometric studies have found favorable internal consistency, factor structure, and concurrent and discriminant validity with diverse community and clinical samples (Brown, et al., 2012; Chorpita, Moffitt, & Gray, 2005; Chorpita, et al., 2000). Cronbach’s alpha for the MDD scale ranged from .80 to .86, and .82 to .86 for the ANX scale across the study period.

Center for Epidemiological Studies-Depression Scale (CESD; Radloff, 1977)—

We used a 22-item modified version of the CESD (mCESD) as a self-report measure of depressive symptoms in adults. The specific and only modification involved the addition of 2 items to address loss of interest and engagement in pleasurable activities, which are core symptoms in the diagnosis of a major depressive episode in the DSM-IV (American Psychiatric Association, 1994) but are not directly reflected in the CESD items. The specific items added were: “I was a lot less interested in most things” and “I was unable to do things I used to enjoy.” The original 20-item CESD has demonstrated sound psychometric properties in clinical and community populations, including samples of women (Knight, Williams, McGee, & Olaman, 1997). Cronbach’s alpha for the mCESD in this sample ranged from .80 to .89 across the study period.

Models ¹

Autoregressive Models—Autoregressive models, also called Markov simplex or univariate simplex models, describe change in a variable over time in terms of the measurement of that variable on previous measurement occasions. Here, we began with a

¹Combined latent curve and autoregressive models (Bollen & Curran, 2004; Curran & Bollen, 2001) were also fit. The intercept-only model for child depression had excellent fit $\chi^2(4) = 1.66, p = .798$; TLI= 1.01; CFI= 1.00; RMSEA= .00; BIC= 4980.01. However, the linear growth model did not result in improved fit, $\chi^2(2) = .90, p = .639$; TLI= 1.01; CFI= 1.00; RMSEA= .00; BIC= 4990.18. Furthermore, the growth factors were not significant indicating that there was not significant change in symptoms over time. We felt that the absence of a significant slope that the intercept model represented the more parsimonious model. The same was true for the latent curve model of maternal depressive symptoms. The intercept only model resulted in a more parsimonious fit, $\chi^2(8) = 1.70, p = .982$; TLI= 1.03; CFI= 1.00; RMSEA= .00; BIC= 6045.48, than the growth model, $\chi^2(5) = .93, p = .968$; TLI= 1.03; CFI= 1.00; RMSEA= .00; BIC= 6060.84, and again the slope factors were not significant. Thus, we were not able to combine the latent curve with the autoregressive model and only the results of the autoregressive models are presented.

first-order autoregressive model that only accounts for the measurement immediately prior to the one of interest. In other words, adolescent depression scores at time t are predicted only by adolescent depression at time $t-1$. Consistent with Curran and Bollen (2001), adolescent and maternal depressive symptoms were first modeled using separate autoregressive models to examine each model's ability to adequately describe depression symptom scores. Depression scores at each time point (e.g., T2) were regressed onto depression scores from the previous wave (e.g., T1) to determine the extent to which adolescent depression scores are predicted by the adolescents' depression scores from the previous year, and the extent to which maternal depression scores are predicted by the mothers' depression scores the previous year. We used the fit statistics described below to identify the best fitting adolescent depression and maternal depression models before combining the models into a single cross-lagged model.

Autoregressive cross-lagged models—The multivariate case of the autoregressive model is the autoregressive cross-lagged model. This model combines the separate univariate models and allows the examination of the relationship between the two processes. The autoregressive models assume that the autoregressive and cross-lagged effects are the same for each person in the sample. For this study, adolescent depression scores at time t were regressed on adolescent depression at $t-1$ and mother's depression at $t-1$, and maternal depression scores at time t were regressed on maternal depression scores at time $t-1$ and adolescent depression scores at $t-1$. This model examines the bi-directional transactional model of maternal and adolescent depressive symptoms by testing the extent to which maternal depression scores are predicted by adolescent depression scores the previous year above and beyond the maternal depression scores the previous year, and the extent to which adolescent depression scores are predicted by maternal depression scores above and beyond adolescent depression scores the previous year. Fit statistics were used to identify the best fitting cross-lagged model before the covariates age, gender, and ethnicity were added to the model. This process was repeated with adolescent anxiety scores to test the exploratory hypothesis that maternal depression would also influence adolescent anxiety.

Data Analysis

Analyses were conducted in MPlus, version 6 (Muthen & Muthen, 1998–2010) using maximum likelihood estimation with standard errors that are robust to non-normally distributed observations, as data were slightly positively skewed (skewness ranged from .56 to 1.39 for RCADS, and 1.06 to 1.43 for mCESD) and kurtotic (kurtosis values ranged from .04 to 3.24 for RCADS, and .529 to 2.52 for mCESD). Modification indices were examined after each iteration to determine the best-fitting models. Only pathways that represented theoretically meaningful relationships or that were supported by prior empirical evidence were added to the models to reduce the likelihood of non-generalizable results. Best fitting models were selected and then the covariates of adolescent age, gender, and ethnicity were added to determine which model best described mother and adolescent depression over time. In addition to χ^2 , fit indices were used to evaluate model fit according to the following criteria: the root mean squared error of approximation (RMSEA) at or below 0.08 (Hu & Bentler, 1999), and Tucker–Lewis index (TLI; Tucker & Lewis, 1973) and comparative fit index (CFI) of 0.95 or higher (Hu & Bentler, 1999). The Bayesian

Information Criterion (BIC; Schwarz, 1978) was also used to compare the relative fit of the models. Models with a lower value of the BIC are considered to be a better fitting, more parsimonious, model.

Results

Descriptive Statistics

Missing data analyses in SPSS (version 19) revealed few significant ($p < .05$) missing data patterns such that parents with missing data at T3 reported lower symptoms of depression at T4, ($t = 2.3$; $df = 7.9$), and had children who reported lower symptoms of depression at T3 ($t = 2.6$; $df = 11.5$) and T4 ($t = 2.5$; $df = 14.5$). Children who were missing data at T3 had parents who reported higher symptoms of depression at T4 ($t = 2.7$; $df = 6.1$). Zero-order correlations between demographic and outcome variables are presented in Table 1. Adolescent ethnicity was significantly correlated with income, such that non-EA youth were more likely to be living in households with lower annual income ($r = -.47$, $p < .01$). A small, but significant correlation was also noted between adolescent ethnicity and adolescent depression scores at T4, such that non-EA adolescents were more likely to report higher depression scores ($r = -.14$, $p < .05$). Adolescent gender was associated with adolescent depression scores at T3 and T4 ($r = -.16$, $p < .05$; $r = -.14$, $p < .05$, respectively), such that females reported slightly higher depression scores. Examining within-time correlations, maternal depression scores at T1 were not associated with adolescent depression scores at T1, nor was there a relationship between maternal depression scores at T2 and adolescent depression at T2. However, maternal depression and adolescent depression were associated at T3 and T4 ($r = .22$, $p < .05$; $r = .21$, $p < .05$, respectively). As noted in the Measures section, the CESD scores used in the analyses reflect the use of a modified 22-item scale; the M 's and SD 's when limited to the original 20 item version are T1 ($M = 9.85$; $SD = 8.36$), T2 ($M = 9.49$; $SD = 8.26$), T3 ($M = 9.41$; $SD = 8.12$), and T4 ($M = 8.96$; $SD = 7.96$). When comparing the 20-item scores to the cutoff of 16 for the beginning of clinically significant symptoms (Radloff, 1977), the scores here are clearly below that value but also indicate a meaningful range of symptoms with one SD overlapping with this clinical cutoff. The scores for RCADS MDD and ANX from the current sample are below these recommended cutoff scores indicating clinically significant distress reported by Chorpita, Moffitt, and Gray (2005), but indicate a meaningful range of symptoms with overlap at one SD.

Adolescent Depressive Symptoms

Adolescent depression scores across the 4 time points (T1–T4) were first examined using the simplex model in which depression scores were regressed onto the year immediately preceding. This basic simplex model resulted in modest model fit, $\chi^2(3) = 8.69$, $p = .034$; $TLI = .95$; $CFI = .97$; $RMSEA = .09$; $BIC = 4997.50$. Modification indices recommended including additional autoregressive parameters for measurements 1 and 3. Given research demonstrating long-term stability of depressive symptoms in youth over time (Keenan, et al., 2009) we augmented the first-order autoregressive model to include effects from more distant time points based on the suggested modification indices. T3 was regressed onto T2 and T1. This resulted in improved model fit (see Table 2, Figure 1). $\chi^2(2) = 4.22$, $p = .12$; $TLI = .97$; $CFI = .99$; $RMSEA = .07$; $BIC = 4996.02$. Significant residual variance for RCADS

scores remained despite the good model fit, suggesting the need for additional explanatory variables in the model (β 's = .56, .52, and .56, p 's < .001, for T2, T3, and T4, respectively).

Maternal Depressive Symptoms

The basic simplex model for mother's depression scores resulted in poor fit $\chi^2(3) = 29.41$, $p < .001$; TLI = .71; CFI = .85; RMSEA = .19; BIC = 6116.54. As with the youth autoregressive model, modification indices recommended adding additional parameters between T1 and T3, and between T2 and T4. Again, this was consistent with research demonstrating stability of depressive symptoms in mothers (Gross, Shaw, Burwell, & Nagin, 2009), thus these parameters were added to the model. Each resulted in incremental improvement in model fit, with significant improvement in BIC, $\chi^2(1) = 5.05$, $p < .03$; TLI = .86; CFI = .98; RMSEA = .13; BIC = 6087.58 (see Table 2 and Figure 1). This model suggests that depression scores at a given time predict depression scores up to 2 years later. Fit indices yielded mixed results regarding model fit with CFI suggesting good model fit, but TLI and RMSEA suggesting poor model fit. Significant residual variance suggests that there is significant unexplained variance in maternal depression scores (β 's = .69, .57, and .58, p 's < .001, for T2, T3, and T4, respectively) that may be accounted for by adolescent depression scores in the combined model.

Maternal and Adolescent Depressive Symptoms

Autoregressive cross-lagged models—The transactional model of maternal and adolescent depression was evaluated by combining the best fitting adolescent simplex models and mother simplex models above and adding cross-lagged parameters between maternal and adolescent depression scores. The influence of maternal depression on adolescent depression was tested by regressing adolescent depression scores onto mother's depression scores from the previous year. To test for the influence of adolescent depression on maternal depression, cross-lagged parameters were included that regressed maternal depression scores onto adolescent depression scores the previous year, $\chi^2(9) = 31.38$, $p < .001$; TLI = .89; CFI = .96; RMSEA = .10; BIC = 11121.07. Within-time correlations between maternal and adolescent depression scores were not significant, r 's = .04, -.08, .05, .07, p 's > .25, for T1 through T4 respectively. Cross-lagged parameters for adolescent depression scores predicting maternal depression scores were not significant $\beta_{\text{child1-mom2}} = .02$, $SE = .05$, $p = .74$; $\beta_{\text{child2-mom3}} = .03$, $SE = .07$, $p = .648$; $\beta_{\text{child3-mom4}} = .124$, $SE = .07$, $p = .06$.

These parameters were removed from the next model, which included the simplex models above, with first-order cross-lagged parameters for maternal depression scores predicting adolescent depression scores one year later. This model was slightly improved over the previous model, $\chi^2(13) = 35.28$, $p < .001$; TLI = .92; CFI = .96; RMSEA = .08; BIC = 11105.18. As with the univariate models, modification indices recommended the addition of second-order cross-lagged parameters from mother's depression scores to adolescent's depression scores 2 years later. This model fit the data well, $\chi^2(14) = 23.74$, $p = .05$; TLI = .97; CFI = .98; RMSEA = .05; BIC = 11083.51.

Covariates of adolescent age at T1, adolescent gender, and ethnicity were added to the model as covariates to T1 depression scores for mothers and adolescents. This resulted in

improved model fit according to some indices, $\chi^2(31) = 39.98, p < .129$; TLI = .97; CFI = .99; RMSEA = .04. However, BIC was significantly larger (BIC = 11786.73) and all parameter estimates were not significant: $\beta_{\text{age}} = -.06, SE = .07, p = .392$; $\beta_{\text{gender}} = .04, SE = .07, p = .519$; $\beta_{\text{minority}} = -.05, SE = .06, p = .483$, suggesting that these covariates did not add significant explanatory power to the model, and were therefore removed for parsimony².

Therefore, the best fitting model included second-order cross-lagged paths between maternal depression scores and adolescent depression scores two years later (Table 2 and Figure 2). These results suggest that adolescent depression scores were best predicted by the adolescent's depression scores the previous year, as were maternal depression scores, as indicated by the large parameter estimates of the autoregressive portion of the model. Furthermore, maternal depression scores at T1 significantly predicted adolescent depression scores at T2 and T3, above and beyond the adolescent's own previous depression scores. The results indicate that maternal depression scores may continue to exert influence on adolescent depression scores, above and beyond the adolescent's previous depression score, for up to two years. However, adolescent depression scores from the same or previous years did not predict maternal depression scores. The absence of within-time correlations in the cross-lagged model suggest that bivariate correlations that were previously observed at T3 and T4 are likely better explained by the autoregressive parameters and the influence of maternal depression on adolescents.

Maternal Depression and Adolescent Anxiety

The best fitting maternal depression simplex model from the previous analysis was carried forward to the adolescent anxiety model. First, adolescent anxiety symptoms were examined using the simplex model. The basic ANX simplex model resulted in good model fit, $\chi^2(3) = 7.68, p = .05$; TLI = .93; CFI = .97; RMSEA = .08; BIC = 6754.59. As with depression, modification indices recommended including additional autoregressive parameters for measurements 2 and 4 and so T4 was regressed onto T3 and T2. This resulted in improved model fit (see Table 2, Figure 1) $\chi^2(2) = 4.22, p = .12$; TLI = .97; CFI = .99; RMSEA = .07; BIC = 4996.02. Significant residual variance for RCADS scores remained despite the good model fit, suggesting the need for additional explanatory variables in the model (β 's = .62, .67, and .51, p 's < .001, for T2, T3, and T4, respectively).

The transactional model of maternal depression and adolescent anxiety was tested by modeling cross-lagged regressions of adolescent anxiety scores onto mother's depression scores from the previous year, and cross-lagged parameters that regressed maternal depression scores onto adolescent anxiety scores the previous year. This model fit the data poorly $\chi^2(9) = 29.42, p < .001$; TLI = .85; CFI = .95; RMSEA = .09; BIC = 13175.02. Within-time correlations between maternal and adolescent anxiety scores were not significant. All

²The modification index recommended adding gender as a predictor of offspring depressive symptoms at T3, which would be consistent with findings that differences in depression between males and female does not start to emerge until around age 13 (Hankin & Abramson, 2001). However, inclusion of this covariate at T3 did not lead to a significant improvement in model fit and lead to a 400-point increase in BIC (11465.074), indicating the model had significantly less parsimony than the model without covariates. The standardized parameter estimate for gender was significant but quite small, $-.113 (SE = .045), p = .011$. The differences in parameters were also negligible, with most parameters differing by .002 points. The largest parameter difference was approximately .02 points and did not affect nominal significance (RCAD3 ON RCAD1 without gender = 0.180, $p = 0.017$; with gender = 0.199, $p = 0.008$).

cross-lagged parameters for adolescent anxiety scores predicting maternal depression scores were not significant. Only one parameter for maternal depressive symptoms predicting adolescent anxiety symptoms was significant $\beta_{\text{mom2-child3}} = .14$, $SE = .06$, $p = .29$. The modification index recommended adding a second-order cross-lagged parameter from mother T1 to adolescent T3. This was added to the next model, and all nonsignificant parameters were removed. The addition of the second-order parameter predicting adolescent anxiety symptoms $\beta_{\text{mom1-child3}} = .27$, $SE = .08$, $p < .001$, resulted in the first order parameter dropping from significance. This model yielded good fit $\chi^2(12) = 18.24$, $p = .11$; $TLI = .96$; $CFI = .98$; $RMSEA = .05$; $BIC = 12198.54$. Age and gender were added to the model, but resulted in degradation of the model and parameter estimates were not significant, $BIC = 12543.79$. The best fitting maternal depression and adolescent anxiety model (Table 2) revealed only one association between maternal depressive symptoms at T1 predicting adolescent anxiety at T3.

Discussion

This is the first study, to our knowledge, to examine the transactional effects of maternal and adolescent depressive and anxiety symptoms over time using the autoregressive cross-lagged design, a powerful method for exploring the temporal association of two variables over time. Studying predictors of depression during this time period is particularly important given the continuity of depression from adolescence into young adulthood (Lewinsohn, Rohde, Klein, & Seeley, 1999) and adulthood (Weissman, et al., 2006). There were five key findings from the present study that will be discussed in turn.

First, maternal depression scores assessed at T1 significantly predicted adolescent depression scores one and two years later (T2 and T3). These findings directly demonstrate the long-lasting influence of maternal depressive symptoms on adolescent depressive symptoms as significant effects were observed up to 2 years later, above and beyond the effects of previous adolescent and maternal depression scores. This finding is consistent with previous research suggesting that adolescents of depressed mothers are more likely than adolescents of non-depressed mothers to report depression and other psychopathology during adolescence and adulthood (Billings & Moos, 1985; Weissman, et al., 2006).

It should be noted that although the models investigated here fit the data well, there remained significant unexplained variance suggesting maternal depression accounts for only a portion of the variance in adolescent depression. More research is needed to investigate the ongoing interactions between mother and youth reports of psychiatric difficulties into adolescence, and to determine how psychiatric risk factors develop and are sustained during this developmental period, as the relationship between mother and offspring depression is theorized to develop under complex pathways. For example, the heritable liability for depression interacts with environmental factors, such that offspring may be exposed to more environmental stress as a result of parental depression (Silberg, Maes, & Eaves, 2010), and that offspring at genetic risk for depression are also more sensitive to stressful life events in general, thereby increasing susceptibility to mood disturbance (Eaves, Silberg, & Erkanli, 2003). Although the autoregressive parameters in the model account for the influence of preexisting depression that may have been influenced by genetic and environmental factors

that may have been set in motion in early childhood, it does not necessarily account for ongoing interaction of genetic vulnerability and environmental factors. Related lines of research have found support for specific cognitive processes, such as rumination (Flanckbaum, et al., 2011) and depressive inferential styles (Abela, Skitch, Adams, & Hankin, 2006; Abela, et al., 2009), as well gene x environment interactions (Hankin, et al., 2011) that may account for the associations between maternal and offspring depression.

Second, maternal depression scores were unrelated to adolescent depression scores assessed at the same time point after accounting for the adolescents' previous depression scores. Although significant bivariate correlations were observed at T3 and T4, these pathways were not significant in the autoregressive models. There are several potential explanations for this finding. One potential explanation is that the exposure of the adolescent to maternal depression at any point may contribute to the adolescent's risk for depression irrespective of timing of maternal depression. This idea has found support in previous literature such that the presence of even a very brief period of maternal major depression or prolonged mild depression predicts risk for depression by adolescence (Hammen & Brennan, 2003). Another potential explanation is that there may be additional mediators of the relationship between maternal depression and adolescent depression that account for the lack of concurrent predictive utility of maternal depression for predicting adolescent depression. For example, parenting style is one factor that is affected by maternal depression that may lead to the time-lagged relationship between maternal depression and adolescent depression (for review, Lovejoy, et al., 2000).

Third, we tested but did not find a reciprocal relationship in which adolescent depression scores predicted maternal depression scores. This finding is in contrast with other studies examining transactional theories of maternal depression and child behavior problems (Bagner, et al., 2012; Gross, et al., 2009; Gross, Shaw, Moilanen, Dishion, & Wilson, 2008), including the only other study using an autoregressive model (Bagner, et al., 2012). Three hypotheses come to mind that warrant further examination. First, the association between child behavior problems and maternal depression in previous studies may have been inflated by the use of parent-report (De Los Reyes & Kazdin, 2005). One possibility is that depressive symptoms assessed in this study are less observable to parents than behaviors such as anxious avoidance or conduct problems assessed by Bagner and colleagues. Indeed, previous research has found that discrepancies between child and parent reports are higher for internalizing symptoms compared to observable behavior problems (see De Los Reyes & Kazdin, 2005 for review) and that the use of youth self-report of depression symptoms leads to increased effect sizes for the relationship between parenting and child depression (McLeod, Weisz, & Wood, 2007). The use of youth self-report in this study may more accurately assess the relationship between maternal and child depressive symptoms. Second, given that Bagner (2012) examined broad child internalizing and externalizing behavior problems rather than depressive symptoms specifically, another hypothesis is that the findings do not generalize to child depressive symptoms specifically. Indeed Gross and colleagues (2009) examined the transactional model of child behavior and maternal depression and found that the transactional model was significant for child externalizing, but not internalizing, symptoms. Third, Bagner and colleagues' (Bagner, et al., 2012) study focused on children between the ages of four and seven, and it is possible that the

transactional processes between mothers and youth change as youth enter adolescence, since youth tend to spend less time with parents and more time with peers beginning in adolescence (Larson & Richards, 1991; Larson, et al., 1996). Although parents continue to exert influence on the development of their offspring throughout adolescence, research suggests that this influence may be indirect (Brown, et al., 1993). Longitudinal studies that bridge these developmental periods are needed to directly examine this possibility.

Fourth, depression scores for both mothers and adolescents were fairly stable across time. This finding is consistent with the line of literature suggesting the stability of depression both among adolescents and adults (Gross, et al., 2009; Keenan, et al., 2009). Depression during adolescence predicts continuation of depression into young adulthood and depressed adolescents also have high rates of nonaffective disorders during young adulthood (Lewinsohn, et al., 1999). These data suggest the need for continued research in the trajectories, outcomes, and treatment of juvenile onset depression.

Lastly, we tested whether the relationship between maternal depressive symptoms and offspring depressive symptoms generalized to offspring anxiety. Our analyses revealed very weak cross-lagged associations between maternal depressive symptoms and offspring anxiety symptoms with only maternal depressive symptoms at T1 predicting adolescent anxiety symptoms at T3. Including reciprocal pathways from offspring anxiety to maternal depressive symptoms did not lead to significant improvement in model fit. These results suggest that there may be specificity of the influence of maternal depression on multiple child outcomes. As previously reviewed, the majority of transactional studies have found associations between maternal depression and child behavior problems (Combs-Ronto, Olson, Lunkenheimer, & Sameroff, 2009; Curran, et al., 1997; Feng, et al., 2009; Sameroff, 2009), yet we failed to find evidence of transactional processes for offspring depression or anxiety. Previous studies have reported inconsistent associations between maternal depression and offspring anxiety. For example, Hammen and Brennan (2003) and Brennan and colleagues (2002) reported no associations between maternal depression and nondepressive offspring disorders including anxiety. In their series of longitudinal studies, Weissman and colleagues did not find a significant association between maternal depression and offspring anxiety disorders in offspring aged 6–23 (1987), yet they found significant relationships between maternal depression and offspring anxiety when participants were assessed 10 and 20 years later (1997, 2006). One possibility is that the pathway between maternal depression and offspring anxiety stretches over a longer period of time. Some have speculated that anxiety and depression may not represent distinct disorders in childhood versus a single internalizing construct that differentiates into separate disorders in later adolescence and adulthood (Seligman & Ollendick, 1998). Others have found that anxiety often precedes depression and have drawn causal links between the two in a developmental framework (Brady & Kendall, 1992). The results of the present study suggest that, at least by adolescence, there may indeed be meaningful distinctions between anxiety and depression, and the risk factors for each disorder may not completely overlap.

The present research has several limitations. First, although the study design is prospective, the follow-up period examined here was limited to adolescence and future research is needed to examine trajectories of mother and adolescent depressive symptoms across

multiple developmental stages (e.g., childhood, adolescence, and emerging adulthood) to gain a more complete picture of how these processes develop over time. Second, we relied on self-reports of depressive symptoms rather than clinical interviews and did not include measures capturing past history or chronicity of depression. Thus, we were unable to examine whether specific patterns of chronicity or remittance/recurrence of maternal depression was related to adolescent depression. Third, we examined depressive symptoms in a community sample reporting a full range of depressive symptoms versus a clinically referred sample. While we believe it is important to understand these processes in the general population, it may be that the transactional influence of child depressive symptoms on maternal depressive symptoms emerges only in clinical populations. These represent important areas for future research. Along these lines, research examining specific depression trajectories, such as the effects of maternal depression remittance on adolescent depression, is a next logical step to further elucidate the relationship between maternal and adolescent depression.

The autoregressive cross-lagged model is not without its limitations. Autoregressive cross-lagged models are only one of several methods for examining relationships between variables over time. We selected this model because it allows us to examine the extent to which adolescent depressive symptoms are predicted by the mother's earlier report of depressive symptoms, above and beyond the adolescent's previous report of depressive symptoms. Although we can extend the model to include parameters from additional years (e.g., $t-1$ and $t-2$), this model does not model growth across all time periods, nor does it consider individual differences in the trajectory of depression over time. Combined autoregressive and latent growth curve models have the benefit of modeling time-specific changes in depressive symptoms while taking into account individual growth trajectories (Bollen & Curran, 2004). We were unable to examine these models due to absence of significant latent growth factors¹, potentially owing to restricted range of the non-clinical community sample. It is possible that including participants with clinical levels of depression may provide sufficient variability within and across time to model latent growth trajectories. Future studies are needed to examine predictors of individual differences in depression trajectories using latent growth modeling procedures as this may provide insight into important questions about how the overall course of maternal depression (e.g., improvement, decline, or stability) affects the course of youth depression.

Implications

These findings have important implications for future clinical research examining the effects of treatment for maternal depression on adolescent depression and anxiety. Previous research has found that improvements in maternal depression following treatment are predictive of improvements in childhood internalizing and externalizing problems during early childhood (Shaw, et al., 2009); however, little research has examined adolescent depression and anxiety in the age group examined here. Emerging research has demonstrated positive effects of treating maternal depression on offspring global symptoms (Wickramaratne, et al., 2011), but not depression specifically. The finding that maternal depression did not have the same effect on adolescent anxiety as depression suggests the need to assess for specific symptoms as meaningful change may be lost in assessments of

global symptoms. Examining the impact of treatment for maternal depression on child depression during late childhood and early adolescence is an important avenue for future research considering the escalation in depression rates during these ages. Furthermore, the time lag observed here suggests that outcomes may need to be followed for several years to adequately assess treatment effects in the offspring of depressed mothers.

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Figure 1.

Adolescent and maternal autoregressive (simplex) models. Adolescent depression and anxiety and maternal depression modeled separately at each time point (T) is predicted by depression scores the previous year. Standardized regression estimates (and standard errors) are presented. All paths were significant ($p < .05$).

Figure 2.

Best-fitting autoregressive cross-lagged model examining the influence of maternal depression on adolescent depression. Depression assessed at 1-year intervals from time 1 (T1) to T4. Significant paths ($p < .05$) are represented by solid arrows. Solid lines represent significant standardized regression estimates (with standard errors). Broken arrows represent paths that were included in the model but were not significant.

Table 1

Descriptive statistics and Pearson correlations between variables.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	M (SD)
1. Child Age	-															12.07 (.91)
2. Child Gender	.148*	-														43.7% female
3. Minority Status	.086	-.038	-													49.1% EA
4. Income	.007	.085	-.459**	-												\$96,191 (\$54,440)
5. A-DEP 1	-.060	.029	-.057	-.039	-											6.12 (4.27)
6. A-DEP 2	-.095	-.053	-.029	-.054	.666**	-										5.95 (4.55)
7. A-DEP 3	-.095	-.161*	-.119	.036	.545**	.678**	-									6.13 (4.59)
8. A-DEP 4	-.126	-.141*	-.142*	.105	.479**	.497**	.660**	-								6.06 (3.84)
9. A-ANX 1	-.076	-.013	.021	-.070	.758**	.527**	.396**	.330**	-							22.41 (13.38)
10. A-ANX 2	-.071	-.189**	-.032	.023	.510**	.732**	.531**	.424**	.602**	-						20.60 (13.02)
11. A-ANX 3	-.117	-.215**	-.056	.001	.375**	.453**	.718**	.509**	.438**	.575**	-					19.92 (12.70)
12. A-ANX 4	-.016	-.240**	-.106	.155*	.355**	.378**	.468**	.689**	.408**	.564**	.663**	-				18.81 (11.65)
13. M-DEP 1	-.099	-.065	.057	-.051	.037	.127	.283**	.172*	.042	.134*	.337**	.183*	-			10.46 (9.11)
14. M-DEP 1	-.154*	-.071	.024	-.095	.036	.039	.142*	.191**	.037	.064	.172*	.164*	.563**	-		10.11 (9.07)
15. M-DEP 1	-.009	-.025	.043	-.092	.138*	.092	.219**	.090	.163*	.142*	.231**	.084	.537**	.615**	-	10.09 (9.96)
16. M-DEP 1	-.003	-.037	.006	-.096	.068	.156*	.230**	.207**	.075	.193**	.190**	.198**	.504**	.583**	.608**	9.97 (8.95)

* Correlation is significant at the 0.05 level (2-tailed).

** Correlation is significant at the 0.01 level (2-tailed).

Note. Child ethnicity coded 0=European-American (EA) or 1=non-EA. Child Gender coded as 0=female; 1=male. A-DEP = adolescent depressive symptoms; A-ANX= adolescent anxiety symptoms; M-DEP = maternal depressive symptoms.

Table 2
Autoregressive models for Maternal Depression, Adolescent Depression, and Adolescent Anxiety

	χ^2	df	p	CFI	TLI	RMSEA (90% CI)	BIC	fp
Depression								
Child Simplex ^a	4.22	2	0.121	0.99	0.97	0.07 (.00 – .15)	4996.02	12
Mother Simplex ^b	5.05	1	0.025	0.98	0.86	0.13 (.04 – .24)	6087.58	13
Cross-lagged, Full ^c	19.79	11	.048	.98	.96	.06 (.005 – .09)	11094.87	33
Cross-lagged, Mother → Child ^d	23.74	14	.049	.98	.97	.05 (.003 – .09)	11083.51	30
Anxiety								
Child simplex	2.46	2	0.292	0.99	0.99	0.03 (.00 – .14)	6748.11	10
Cross-lagged, Full ^e	29.42	9	<.001	0.95	0.85	0.094	12220.77	28
Cross-lagged, Mother1 → Child3 ^f	18.24	12	0.109	0.98	0.96	0.05 (.00 – .90)	12198.54	27

Notes. fp= number of free parameters.

^a Child Simplex model includes additional autoregression between time (T)3 and T1 child depression.

^b Mother Simplex model includes additional autoregression between T3 and T1, and T4 and T2.

^c Cross-lagged, Full model combines child and mother simplex models and includes first and second order cross-lagged parameters from mother to child, and first order parameters from child to mother.

^d Cross-lagged, Child → Mother model combines child and mother simplex models and includes only first and second order cross-lagged parameters from mother to child.

^e Cross-lagged, Full model for anxiety combines child and mother simplex models and includes first order cross-lagged parameters from mother to child, and first order parameters from child to mother.

^f Cross-lagged, Mother1 → Child3 for anxiety model combines child and mother simplex models and includes only one significant cross-lagged parameters from mother at T1 to child at T3.