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Concurrent Trajectories of Change in Adolescent and Maternal Depressive Symptoms in the TORDIA Study

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Abstract

Depression has a heightened prevalence in adolescence, with approximately 15 % of adolescents experiencing a major depressive episode by age 18. Depression in adolescence also poses a risk for future distress and impairment. Despite treatment advances, many adolescents relapse after initial remission. Family context may be an important factor in the developmental trajectory of adolescent depression, and thus in enhancing treatment. This study examined concurrent change over time in adolescent and maternal depressive symptoms in the context of the Treatment of Resistant Depression in Adolescents study. Participants were 334 adolescents (mean age: 16; SD: 1.6; 70 % female, 84 % Caucasian), and their mothers ($n = 241$). All adolescents were clinically depressed when they entered the study and had received previous selective serotonin reuptake inhibitor (SSRI) treatment. Adolescents received acute treatment for 12 weeks and additional treatment for 12 more weeks. Adolescent depression and suicidal ideation were assessed at 0, 6, 12, 24, 48 and 72 weeks, while maternal depressive symptoms were assessed at 0, 12, 24, 48 and 72 weeks. Latent basis growth curve analyses showed a significant correlation over 72 weeks between trajectories of maternal and adolescent depressive symptoms, supporting the hypothesis of concurrent patterns of change in these variables. The trajectories were correlated more strongly in a subsample that included only dyads in which mothers reported at least one depressive symptom at baseline. Results did not show a correlation between trajectories of maternal depressive symptoms and adolescent suicidal ideation. These findings suggest that adolescent and maternal depressive symptoms change in tandem, and that treatment for adolescent depression can benefit the wider family system. Notably, most mothers in this sample had subclinical depressive symptoms. Future research might explore these trajectories in dyads with more severely depressed mothers.

Keywords

Depression; Family system; Maternal depression

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Author Contributions AP and CES developed initial study hypotheses together. AP conducted the literature review, managed and conducted analyses, wrote the first draft of the manuscript, and responded to all edits and revisions. CES edited and helped revise the manuscript. TC provided critical guidance on the statistical analyses and provided guidance in writing and revising the Methods and Results sections. KR also provided edits on an earlier draft of the manuscript. All authors read and approved the final manuscript.

Introduction

Depression is a serious and common public health problem that poses a heightened risk in adolescence. The incidence of major depression increases steadily throughout the adolescent years, with lifetime prevalence rates nearly doubling from ages 13–14 (8.4 %) to ages 17–18 (15.4 %) (Merikangas et al. 2010). Approximately 1 in 20 teenagers suffer from major depressive disorder (MDD) at any given time (Costello et al. 2006). Beginning in early adolescence (i.e., the onset of puberty), girls' rates of depression are higher than those of boys (e.g., Essau et al. 2010), with the most dramatic gender difference occurring during ages 15–18 (Hankin et al. 1998). Depression in adolescence predicts greater risk for recurrent depression in adulthood, as well as for anxiety, substance abuse, suicide, educational underachievement and early parenthood (e.g., Fergusson and Woodward 2002). Thus, effective and early treatment for depression is vital. Despite clear advances in the treatment of adolescent depression, between one-fifth (22 %) (Clarke et al. 1999) and one-half (46.6 %) (Curry et al. 2011) of adolescents who initially recover from depression relapse within 2 years. An enhanced understanding of the trajectories of adolescent depression and of factors associated with these trajectories, especially in the context of treatment, is needed to inform and improve efforts to treat depressed youth.

Developmental Trajectories of Adolescent Depressive Symptoms

The stressful transition to adolescence carries an array of physical, psychological and social changes, and in turn is associated with an increase in depression, which levels off by the end of adolescence (Hankin 2006). Within this overall pattern, however, exist distinct developmental trajectories of depressive symptoms. In the National Longitudinal Study of Adolescent Health (a national sample of more than 20,000 youth), distinct trajectories were defined by chronically high, increasing, decreasing and consistently low levels of depressive symptoms (Wickrama et al. 2009). These groups were, in turn, associated with differences in physical health and socioeconomic characteristics (Wickrama et al. 2009) and risky behavior in young adulthood (Wickrama and Wickrama 2010). Family interactions may be another factor that influences trajectories of adolescent depressive symptoms, especially if parents are facing their own mental health struggles.

Effect of Parental Mental Health on Adolescent Depression Trajectories

Parental psychopathology is a well-established risk factor for adolescent mental health problems and functional impairment (e.g., Lewinsohn et al. 2005), especially in family environments where other stressors are present (Hammen and Brennan 2003). Children of depressed parents have an earlier onset of mood disorders (Weissman et al. 1987) and are at increased risk for depression, anxiety disorders, substance dependence and social impairment in adolescence (Weissman et al. 1997) and into adulthood (Weissman et al. 2006). They are also at increased risk for suicidal behavior (King et al. 2010), particularly if a parent has completed or attempted suicide (Christiansen et al. 2011). Though the relationship between parental and youth depression can be explained in part by genetic transmission (Eley 1999), environmental or familial stressors such as parental mental health also play a significant role (Eley et al. 2004).

There is evidence that parental depression, and that of mothers in particular, affects the developmental trajectory of adolescent psychosocial adjustment, though evidence is more robust in supporting an influence on initial *levels* of adolescent symptoms, not on the *growth* (i.e., change) of symptoms over time. For example, in one 6-year longitudinal study, children of depressed mothers had higher initial levels of depression, but their trajectories of depressive symptoms, stressors and attributional style did not differ from those of youth with non-depressed mothers (Garber et al. 2002). In a study that followed adolescents and

families for 12 years (from ages 5 to 17), maternal depression predicted internalizing symptoms in adolescent boys and girls at age 17, but affected the slope (i.e., rate of change) of internalizing symptoms only in girls (Leve et al. 2005). This study did not examine the impact on adolescent depression in particular. A recent longitudinal study provides some evidence for related symptom change over time, suggesting that escalation in maternal anxiety and depression over 5 years is associated with a concurrent increase in child/adolescent mental health problems (Fatori et al. 2013). Notably, this study also explored the impact on a broad dependent variable, rather than on adolescent depression specifically.

Subclinical maternal depressive symptoms also appear to play an important role in adolescent adjustment and thus deserve study. A large-scale study of mother–child dyads revealed five trajectories of maternal depression over the first 12 years of the children’s lives (never depressed; stable subclinical; early-decreasing; moderately elevated; chronic) (Campbell et al. 2009). When these children were assessed at age 15, those whose mothers were in the *stable subclinical*, *moderately elevated* or *chronic* trajectories reported more internalizing and externalizing symptoms than did children of *never-depressed* mothers. This finding is not surprising given that subclinical depressive symptoms are associated with substantial distress and impairment, though these outcomes are less severe than those of full-blown clinical depression (e.g., Cuijpers et al. 2004; Howland et al. 2008). Subclinical depression also incurs a risk for later development of major depression, both in the individual experiencing the symptoms (Fogel et al. 2006) and in that individual’s relatives (Lewinsohn et al. 2003). It follows, therefore, that maternal subclinical depression also could play a pivotal role in a family system.

Inter-relationship Between Maternal and Adolescent Depressive Symptoms

Family systems theory posits, broadly, that a change in any part of the family environment affects each individual in the family (e.g., Chabot 2011). The family is an interdependent system that is organized to change and meet challenges during difficult transitions, and it can become stuck in a transition if individuals or the system are unable to adapt (Gerson 1995). Adolescence, characterized by teenagers’ search for autonomy and parents’ need to adapt to evolving roles in their children’s lives, is a prime example of such a transition. Families with an adolescent or a parent who is coping with depression face an even greater challenge (Campbell et al. 2009).

From this family systems perspective, maternal and adolescent depressive symptoms are likely to have interconnected trajectories. For example, if a depressed parent has difficulty adapting to a changing family environment during a child’s adolescence, the adolescent may perceive less parental support or love and be more prone to develop depression or other mental health difficulties (e.g., Stice et al. 2004). Similarly, as an adolescent struggles to manage the emotional, physical and social changes that accompany the transition to adolescence, and especially if the adolescent is genetically predisposed to depression, he/she may experience depressive symptoms (e.g., sullenness, irritability, diminished energy) that frustrate or dishearten a parent and negatively affect that parent’s mental health. Indeed, adolescents with depression show an escalation in dysphoric behavior during conflictual interactions with their parents, in comparison to a stable level of dysphoric behaviors in non-depressed adolescents’ interactions (Sheeber et al. 2012). As these parent- and adolescent-driven processes occur simultaneously, they can exacerbate each other in a self-perpetuating cycle.

Indeed, a number of community-based longitudinal research studies have found evidence to suggest that parental and youth mental health affect one another. Some studies have found that maternal depression predicts depression among offspring (e.g., Ge et al. 1995; Hammen; Kouros and Garber 2010). Others suggest that higher levels of adolescent

depression predict greater parental depression (Ge et al. 1995), though this has pattern has not consistently been found (Forehand and McCombs 1988). Collectively, theory and much community-based research suggest parent and adolescent depression are interrelated over the course of time (Elgar et al. 2004; Hughes and Gullone 2008), though the precise nature of the relationship is not understood fully.

Overall, an association exists between maternal and offspring mental health, and depressive symptoms in particular, with both empirical evidence and family systems theory suggesting that these two phenomena are interrelated over the course of time. Still, the relationship between *change* in these two variables has not been studied longitudinally. This leaves a question about the relationship of these trajectories, especially as one (or both) members of the mother–child relationship participate in treatment to address their depressive symptoms.

Maternal Depression in the Context of Treatment for Adolescent Depression

A youth depression treatment study—in which change in adolescent depression is *expected*—provides a unique opportunity to examine the degree to which parental and adolescent depressive symptoms change in tandem over time. To date, parental depression primarily has been studied as a predictor or moderator of adolescent treatment outcomes, with mixed results. For example, in a study of fluoxetine treatment for youth ages 7–18 (Kennard et al. 2008), children with more (vs. less) depressed mothers at baseline ended treatment with a higher level of depressive symptoms (Kennard et al. 2008). Further, youth with mothers who reported improvement in depressive symptoms (about half) over the course of their children's treatment had higher remission rates relative to those with mothers who reported no change or an increase in depression. In an earlier randomized controlled adolescent depression study, Brent et al. (1998) found that maternal depression did not predict adolescent outcome in the sample as a whole at 12 weeks, but the presence of self-reported maternal depression symptoms did moderate outcome. Specifically, CBT was more effective than the other two psychosocial treatments, but only in the absence of maternal depression. At 2-year follow-up, however, maternal depression did not influence outcomes in the same sample (Birmaher et al. 2000). Similarly, self-reported maternal depressive symptoms did not predict or moderate outcomes in the Treatment for Adolescent Depression (TADS) project, a multi-site randomized controlled trial of cognitive-behavioral and psychopharmacological treatment for depressed adolescents (Curry et al. 2006).

In sum, data are inconclusive on the role of maternal depression as a predictor or moderator of adolescent treatment outcomes. Moreover, researchers have not explored the role of *ongoing* maternal depressive symptoms in adolescent symptom change. The present study sought to explore the relationship between maternal and adolescent depressive symptoms during and after the adolescents' participation in treatment for depression.

The Present Study: Research Questions

The present study examined maternal depressive symptoms as they relate to adolescent depressive symptoms and suicidal ideation in the Treatment of selective serotonin reuptake inhibitor (SSRI) Resistant Depression in Adolescents study (TORDIA; Brent et al. 2008), an NIMH-funded multi-site randomized clinical trial. The current study builds upon prior research by seeking not only to examine the influence of maternal depression at baseline on adolescent depressive symptoms, but also to examine concurrent growth trajectories of adolescent and maternal depressive symptoms over the course of 72 weeks. We were interested in exploring the trajectories of adolescent symptom change as they relate longitudinally (during and after treatment) to maternal symptom change. Given the documented associations between maternal and adolescent mental health in general, we hypothesized that trajectories of symptom change would also show an association. We also

hypothesized that this association would be strongest in mothers with more depressive symptoms. Such a finding would clarify the ongoing role of maternal depressive symptoms in the course of adolescent depression treatment.

We hypothesized that, consistent with past research, initial levels of maternal depression and adolescent depression would be significantly correlated with each other (Hypothesis 1a), and that this relationship would persist over time, such that the trajectories of change between maternal depressive symptoms and adolescent depressive symptoms would be significantly associated with each other throughout the course of treatment and follow-up periods. In other words, the slopes of these growth curves would be significantly correlated (Hypothesis 1b). Lastly, we hypothesized that these effects would be most pronounced in the subsample of dyads in which mothers reported at least one depressive symptom at baseline (Hypothesis 1c), given that these mothers might be more vulnerable to the volatility (in either direction) of their children's depressive symptoms.

Similarly, we also hypothesized that initial levels of maternal depressive symptoms and adolescent suicidal ideation would be significantly correlated with each other (Hypothesis 2a), and that trajectories of change between maternal depressive symptoms and adolescent suicidal ideation would be significantly associated with each other throughout the course of treatment and follow-up periods. In other words, the slopes of these growth curves would be significantly correlated (Hypothesis 2b). Here, too, we hypothesized that effects would be stronger in the subsample including mothers who reported at least one depressive symptom at baseline (Hypothesis 2c).

Method

An overview of participants and study procedures is described below. See Brent et al. (2008) for additional details of original study procedures. TORDIA Dataset Version 1 was used in this study (Data Use Certificate Version 4_1).

Participants

Participants were 334 adolescents (ages 12–18) who received treatment for major depression through the TORDIA study across one of six study sites (Brent et al. 2008), and 241 of their mothers, who were followed over the course of 16 months (72 weeks). Data from fathers ($n = 64$ at baseline) or other guardians ($n = 36$) who completed study assessments were not included in study analyses because research suggests that maternal factors may exert a greater influence on offspring mental health than do paternal factors (Connell and Goodman 2002; Hughes and Gullone 2008) and there was not ample power to examine these groups separately.

Inclusion criteria for TORDIA included a clinically significant depression [Child Depression Rating Scale-Revised (CDRS-R) score ≥ 40 and Clinical Global Impression-Severity (CGI-S) Subscale ≥ 4] despite having received SSRI treatment for at least 8 weeks. Adolescents were excluded if they had undergone two or more adequate trials of an SSRI, or had shown non-response to venlafaxine or to cognitive-behavioral therapy (CBT). Among other exclusion criteria were diagnoses of bipolar disorder, psychosis, eating disorder, substance abuse or dependence, and hypertension. Females were excluded if they were pregnant, breastfeeding, or not reliably using contraception.

The mean age among the 334 adolescent participants was 15.89 years (SD 1.57 years); 70 % were female and 84 % were white (5 % Hispanic/Latino, 5 % biracial, 3 % black, 2 % Asian, 2 % other). The mothers' mean age was 44.23 (SD: 6.01). The median family income was \$61,000 (range: \$0–\$500,000). At study entry, on average, adolescents had moderately

severe and chronic depression (mean CDRS-R: 59, SD: 10; 56 % duration of 2 years or longer). Co-occurring diagnoses were found in 51.7 % of the adolescents, such as anxiety (38.9 %), conduct or oppositional defiant disorders (9.6 %), and attention deficit hyperactivity disorder (ADHD) (16.6 %). Approximately 37 % of youth reported a history of non-suicidal self-injury, 14.9 % suicide attempts, and more than half (58.5 %) clinically significant suicidal ideation.

Treatment

Participants were randomly assigned to one of four conditions in a 2×2 factorial design: change to a second SSRI, change to venlafaxine, change to second SSRI plus CBT, or change to venlafaxine plus CBT. Acute treatment across conditions lasted for 12 weeks. Participants were tapered off their pre-study medications over the course of 2 weeks (Brent et al. 2008). At week 12, responders [those with a Clinical Global Impression-Improvement (CGI-I) rating of ≤ 2 , denoting much or very much improved] continued in their assigned blinded treatment arm, while non-responders or responders who required additional intervention received open treatment (medication and/or CBT) for 12 more weeks. Treatment groups were similar in their baseline demographic, clinical characteristics, and rates of treatment completion (Asarnow et al. 2009).

The TORDIA CBT was delivered in a flexible manner, with modules chosen based on the case conceptualization, as reviewed in biweekly conference calls. The modules targeted cognitive restructuring, behavioral activation, emotion regulation, social skills, and problem solving. Parent-child sessions aimed to improve support, decrease criticism, and improve family communication and problem solving. Adolescents received as many as 15 sessions during the 12-week acute treatment period, 3–6 of which included parents (mean number of total therapy sessions attended = 8.30; SD = 3.46).

Follow-Up Periods

Adolescents completed assessments at 6, 12, 24, 48, and 72 weeks. Mothers completed assessments at 12, 24, 48, and 72 weeks. Of the 334 adolescents who completed baseline assessments, data were available for 292 (87.4 %) at 6 weeks, 287 (85.9 %) at 12 weeks, 259 (77.5 %) at 24 weeks, 196 (58.7 %) at 48 weeks, and 173 (51.8 %) at 72 weeks. Of the 241 mothers who completed baseline assessments, data were available for 214 (89 %) at 12 weeks, 185 (77 %) at 24 weeks, 149 (62 %) at 48 weeks, and 131 (54 %) at 72 weeks.

Measures

Adolescent Depressive Symptoms—Adolescent depressive symptoms were measured by clinician interview with the Clinician Depression Rating Scale-Revised (CDRS-R) (Poznanski et al. 1985; Poznanski and Mokros 1996), a 17-item interview that measures depression severity. A score of 40 or greater is indicative of clinically significant depression. Consistent with past research (e.g., TADS 2004; Wagner 2003), the outcome measure used was the CDRS-R ‘Best Description’ of the adolescent’s total score, which is the highest-severity rating for each item, in the judgment an independent evaluator who has interviewed both parent and adolescent. The CDRS-R has demonstrated good internal consistency (coefficient $\alpha = .85$), inter-rater reliability ($r = .92$), and test-retest ($r = .78$) reliability. In this sample, inter-rater reliability on the CDRS-R was strong (intraclass correlation coefficient: .85; 95 % CI .80–.89; $N = 324$; Brent et al. 2008).

Adolescent Suicidal Ideation—Suicidal ideation in the past month was assessed with the 15-item Suicidal Ideation Questionnaire-JR (SIQ-JR; Reynolds and Mazza 1999). Scores of 31 and higher are indicative of clinically significant suicidal ideation. The SIQ-JR has shown good internal consistency ($\alpha = .91$), test-retest reliability over approximately 3 weeks

($r = .89$), and validity (e.g., differentiated adolescents who had vs. had not attempted suicide in the past year) (Reynolds and Mazza 1999). Internal consistency was high in this sample ($\alpha = .96$).

Maternal Depressive Symptoms—Maternal depressive symptoms were measured with the BDI-II (Beck et al. 1988), a 21-item measure of severity of affective, cognitive and somatic depressive symptoms in the past 2 weeks. The BDI-II has demonstrated high internal consistency (average coefficient $\alpha = .81$) and test–retest reliability ($r = .83$). In this sample, internal consistency of the BDI-II was excellent ($\alpha = .93$) among mothers.

Analysis Plan

Study analyses controlled for gender and CBT treatment condition. Research suggests that girls are more sensitive to interpersonal stressors, including parental depression (e.g., Davies and Windle 1997; Sarigiani et al. 2003) and family conflict (Rudolph 2002; Skeer et al. 2011) than are boys. The treatment condition was collapsed across type of medication and dichotomized into CBT versus no CBT groups, because the medication conditions did not differ from each other in the degree of parental involvement in treatment, but did differ from the CBT condition in this regard. Moreover, treatment response did not differ between the two medication groups, but did differ between groups who did or did not receive CBT, as described in previously published TORDIA reports (Brent et al. 2008; Vitiello et al. 2011).

Bivariate latent basis growth curves were used to examine the relationships between trajectories of maternal depressive symptoms and adolescent symptom improvement. We ran two separate models, each of which examined the growth relationships between maternal depression and one of two adolescent symptom measures (depressive symptoms or suicidal ideation). We also ran the analyses with two different samples: first with the full sample, and second with a sub-sample limited to dyads in which the mother endorsed at least one symptom of depression at baseline (BDI-II > 0). Analysis of bivariate latent growth curves allows for estimation of both latent intercept and latent slope terms for each set of variables. The latent intercepts allowed for estimation of initial statuses (e.g., pre-treatment), and the latent slopes provided measures of growth across time (e.g., change in severity of depressive symptoms). This model assumes that a single trajectory underlies the growth in each variable across all assessment time points (Ferrer and McArdle 2003). The use of a latent basis growth curve model allowed for non-linear growth, which was expected in this sample, given that an acute treatment phase was followed by a longer follow-up phase without study treatment. This was specified in the model by setting the initial (intercept) slope loading to zero and the final loading to 72, with the other loadings being free to vary. The choice of zero as the initial time point and 72 for the final time point allows the other loadings to be interpreted in terms of weeks. Within the same model, two pairs of these growth models were constructed—one for the mother and one for the adolescent. All pairwise correlations were then estimated between initial levels and slopes.

Of primary interest were the correlations between slopes. Specifically, we examined correlations between (1) slope of maternal depressive symptoms (BDI-II) in relation to slope in adolescent depressive symptoms (CDRS-R) and (2) slope of maternal depressive symptoms (BDI-II) in relation to slope in adolescent suicidal ideation (SIQ-JR), controlling for gender and CBT treatment condition. See Fig. 1 for a simplified figure of the maternal and adolescent depression model.

All models were estimated with the Analysis of Moment Structures 19.0 (AMOS; Arbuckle 2010) program, using full information maximum likelihood (FIML) estimation, which uses all data available at each time point to account for missing data. Multiple measures of fit were considered in evaluating model quality, as is generally recommended (Tanaka 1993),

given that different fit measures focus on different components of fit (Browne and Cudeck 1993; Hu and Bentler 1995). These included the comparative fit index (CFI) and root mean square error of approximation (RMSEA). These values usually range from 0 to 1. A value of .90 or greater on CFI is generally considered evidence of a good-fitting models (Bentler and Bonett 1980), whereas smaller values of RMSEA support better-fitting models, with values of .07 or lower indicating good fit (Stieger 2007).

Results

Descriptive Results

Descriptive statistics and zero-order correlations of the measured variables are provided in Table 1. Of particular interest are the high correlations between the same variable at each time point, suggesting that these variables are relatively stable over time. Further, adolescent depressive symptoms and suicidal ideation were strongly correlated with each other across time points, suggesting that, on average, suicidal ideation remains associated with depressive symptoms even as both subside, which may reflect general treatment response. Interestingly, maternal depressive symptoms at baseline were not correlated with levels of adolescent depression or suicidal ideation at any time point, but maternal depressive symptoms at several later points did show significant correlations with several follow-up adolescent measures.

The parameter estimates of the latent basis growth curves (slopes and intercepts) of the relevant variables, as estimated by AMOS, are provided in Tables 2 and 3. The estimated initial level of maternal depressive symptoms (BDI-II) in each model was on the high end of the “minimal depression” range (0–13) (Beck et al. 1996), suggesting that this was not a severely depressed sample of mothers. In fact, only 28 % of mothers (69 of 241) reported baseline depressive symptoms of 14 or higher, denoting at least “mild” depression. An even smaller proportion, 16 % (38 of 241), reported at least moderate-level depressive symptoms, and 6 % (15 of 241) reported severe-level symptoms. The estimated initial levels of adolescent depressive symptoms (CDRS-R) and suicidality (SIQ-JR) were both indicative of clinically elevated levels.

The correlations between slopes and intercepts of all the modeled variables are summarized in Tables 4 and 5. The same maternal depressive variables were modeled with adolescent depressive symptoms and with adolescent suicidal ideation. For maternal depressive symptoms, initial level and slope were significantly and negatively correlated with each other ($r = -.72-.73$; $p < .01$ in both cases), indicating that mothers whose initial depressive symptoms were more severe had a faster rate of decline in symptoms over the course of the study. For adolescent depressive symptoms, level and slope were also negatively correlated, but the relationship was not statistically significant ($r = -.341$, $p = .140$). For adolescent suicidal ideation, however, a strong and significant negative correlation emerged between level and slope ($r = -.765$, $p < .001$), indicating that adolescents whose initial suicidal ideation was higher had a much faster rate of decline in suicidal ideation during the course of the study. Of primary interest was the correlation between slopes of maternal and adolescent symptom trajectories; these results are discussed in more detail in the following section.

Modeled Growth Trajectories

Each of the three variables of interest followed a non-linear pattern of change. All the slopes were negative and significant, indicating that the mean levels of these symptoms declined significantly over the course of treatment and follow-up. Since each measure has a different scale, the exact slope values cannot be compared meaningfully across variables. The *shapes*

of the trajectories, however, can be compared. These patterns are described below and in Table 6, and illustrated in Fig. 2.

Adolescent Depressive Symptoms—The growth curve for adolescent depressive symptoms (CDRS-R) had a significant negative slope. Parameter estimates for the slope at each time point (i.e., latent basis loading) indicated that the steepest mean decline occurred during the initial 12-week acute treatment period. More than half of the total change over 72 weeks occurred in the first 6 weeks, followed by an additional 10 % in weeks 6–12. In fact, the mean CDRS-R score dropped below the level that denotes clinically significant depressive symptoms (i.e., dropped below 40; Poznanski and Mokros 1996) between weeks 6 and 12, suggesting a rapid initial response to treatment. The symptoms continued to decline (though more gradually) between weeks 12 and 24, and then even more gradually in the follow-up periods.

Adolescent Suicidal Ideation—Adolescent self-reported suicidal ideation (SIQ-JR) also showed a significant and non-linear pattern of decline, with the largest drop in the 12-week acute treatment period, though this descent was less extreme than that for adolescent or maternal depressive symptoms. Twice as much change occurred in the first 6 weeks as in the subsequent 6 weeks. Still, levels of suicidal ideation remained in the clinical range (>31 , per Reynolds and Mazza 1999) at 12 weeks, and declined to the sub-clinical range between weeks 12 and 24. The slope flattened somewhat after acute treatment ended at week 12, with the least change occurring between weeks 24 and 48.

Maternal Depressive Symptoms—The slope of the modeled growth trajectory for maternal depressive symptoms (BDI-II) also indicated an overall significant decline in depressive symptoms. More than half of the total decline occurred in the first 12 weeks. Between weeks 12 and 24, the rate of change stayed relatively stable, though symptom levels actually increased slightly. The decline continued gradually in weeks 24–48, and was slowest in weeks 48–72. Notably, at all time points, including baseline, the mean level of maternal depressive symptoms was below the clinical cutoff, even using a conservative cutoff (“mild” depression) (Beck et al. 1996).

Correlations Between Maternal and Adolescent Depressive Symptoms

We hypothesized that (1a) initial levels of maternal depressive symptoms would be significantly correlated with initial levels of adolescent depression, (1b) the trajectory of change in maternal depressive symptoms would correlate significantly with the trajectory of change in adolescent depressive symptoms, and (1c) this relationship would be stronger in dyads in which mothers reported at least one depressive symptom. This hypothesis was supported partially. Counter to hypothesis 1a, initial levels of maternal and adolescent depressive symptoms were not correlated significantly, though results did indicate a trend toward significant positive correlation ($r = .21, p = .07$), after controlling for gender and CBT treatment condition.

Hypothesis 1b, which predicted that change in maternal and adolescent depressive symptoms would be correlated over time, was supported. Results indicated that, when controlling for gender and CBT condition, the slope of maternal symptom change (mothers’ BDI-II self-report) was correlated significantly and positively with the slope of adolescent symptom change (clinician-rated CDRS-R) ($r = .58, p < .05$). The correlation coefficient indicates that there was 33.6 % shared variance in this relationship, suggesting that the rate of decline in maternal depressive symptoms is associated strongly with the rate of decline in adolescent depressive symptoms over the 72 weeks of the study. Fit indices suggested good fit (CFI = .922, RMSEA = .064). As for covariates, results suggested that adolescent males

had lower initial levels of depressive symptoms by 2.81 unstandardized points on the CDRS-R ($p < .05$). None of the other relationships between gender nor CBT condition and initial levels of maternal or adolescent depressive symptoms were significant.

A subsequent analysis using a subsample of participants—only from dyads in which the mothers reported at least one depressive symptom at baseline ($n = 311$ adolescents; 217 mothers)—lent support to hypothesis 1c. In this model, the correlation between the slopes of maternal and adolescent depressive symptom trajectories was slightly stronger than it was in the full sample ($r = .65$, $p < .05$, compared to $r = .58$). Fit indices suggested good fit (CFI = .92, RMSEA = .06). This suggests that in mothers who reported as little as a single depressive symptom at baseline, there was 42.3 % shared variance between maternal and adolescent symptom change. In examining covariates, results again indicated that adolescent males had lower levels of initial depressive symptoms, this time by 3.42 unstandardized points on the CDRS-R ($p < .01$). None of the other relationships with covariates were significant. In this subsample, as in the full sample, the correlation between initial levels of maternal depressive symptoms and adolescent depressive symptoms showed only a trend toward significance ($r = .23$, $p = .09$).

Correlations Between Maternal Depressive Symptoms and Adolescent Suicidal Ideation

We also hypothesized that (2a) maternal depressive symptoms would correlate at baseline with adolescent suicidal ideation, (2b) these two variables would fluctuate together over time; and (2c) this relationship would be more pronounced in a subsample of mothers with at least one self-reported depressive symptom. Hypothesis 2 was not supported. Initial levels of maternal depressive symptoms and adolescent suicidal ideation were not correlated significantly ($r = .08$, $p = .30$). Furthermore, the slope of maternal depressive symptoms was not correlated significantly with the slope of adolescent self-reported suicidal ideation when controlling for gender and CBT condition ($r = .05$, $p = .77$). Fit indices indicated good fit (CFI = .95, RMSEA = .06). This suggests that adolescent suicidal ideation and maternal depressive symptoms do not fluctuate together as adolescents engage in treatment for depression. As for covariates, results did not suggest significant relationships between initial levels of suicidal ideation with either gender or CBT condition. In the model exploring this relationship in the subsample including only mothers reporting any depressive symptoms (BDI-II > 0) at baseline, the fit remained good (CFI = .95, RMSEA = .06) and the correlation of interest remained non-significant ($r = .05$, $p = .79$). No relationships with covariates (gender or CBT condition) were significant in this subsample. Moreover, the correlation between initial levels of maternal depressive symptoms and adolescent suicidal ideation remained non-significant ($r = .07$, $p = .46$).

Discussion

This study explored whether there was concurrent change in maternal depressive symptoms and adolescent symptoms (depressive symptoms and suicidal ideation) in the context of a clinical trial for adolescent depression. While the association between maternal and adolescent mental health symptoms has been examined previously using longitudinal designs in community samples, this is the first study to explore these mutual effects in a sample of adolescents being treated for depression. We found that the trajectories of change between maternal and adolescent depression fluctuated together, but that trajectories of maternal depression and adolescent suicidal ideation did not.

Trajectories of Change in Maternal and Adolescent Depressive Symptoms

Consistent with Hypothesis 1b, there was a significant correlation between the slopes of the maternal and adolescent trajectories of depressive symptom change. This finding was most

robust when the sample was limited to dyads in which mothers reported at least one symptom of depression at baseline (Hypothesis 1c). Whereas some prior research suggests that maternal depressive symptoms at *baseline* affect initial levels of adolescent depression (e.g., Garber et al. 2002) and moderate adolescent treatment outcomes (e.g., Brent et al. 1998; Kennard et al. 2008), the present study builds on this research to suggest that growth curves of adolescent and maternal depressive symptoms are associated significantly with each other over time. Thus, as an adolescent's depression subsides (in this case, presumably through treatment), his or her mother's depressive symptoms also appear to decline. With regard to timing, both growth curves had the sharpest decline in the acute treatment phase, suggesting that the greatest change occurred within these first 12 weeks. Interestingly and counter to hypotheses, baseline levels of maternal and adolescent depressive symptoms showed only a trend toward a significant correlation, in both the full sample and subsample of mothers with at least one depressive symptom. This surprising finding may have resulted from the relatively restricted range of subclinical maternal depressed symptoms reported in the present sample.

Trajectories of Change in Maternal Depressive Symptoms and Adolescent Suicidal Ideation

Contrary to study hypotheses, growth curves of maternal depressive symptoms and adolescent suicidal ideation were not correlated significantly over time. The baseline correlation was also non-significant, which is contrary to previous research that has found that parental mental illness increases the likelihood of adolescent suicidality (e.g., King et al. 2010). In part, this may be because the strongest association with offspring suicidality is that with parental suicidality, rather than with broader parental mental health (e.g., Brent and Mann 2005). This divergence from the pattern of concurrent fluctuations also may be a function of different assessment tools employed to measure the two adolescent outcomes of interest. Adolescent depression may have been captured more accurately by the clinician rated CDRS-R, which took into account parent and adolescent report, in comparison to the self-report measure of suicidal ideation (SIQ-JR). Moreover, these clinicians were blind to treatment condition, and thus likely were biased less by social desirability, depressogenic thought processes, or other factors that may have affected adolescent self-report.

Disparate findings between adolescent depressive symptoms and suicidal ideation also may be explained by research suggesting that depression and suicidal thoughts are separate, albeit related, constructs with distinct developmental pathways and responses to treatment (Linehan 1997). For example, one study found that cognitive-behavioral therapy was associated with a significant reduction in self-injurious and suicidal behavior in comparison to treatment-as-usual, but was no more effective than the control group in lowering depression or hopelessness (Linehan et al. 1991). Other studies have shown a similar disconnect between improvement in depression and reduction of suicidal behavior (e.g., Gibbons et al. 2012; Wood et al. 2001). In fact, in a meta-analysis of treatment for adolescent depression, the average effect size for change in suicidality (across six studies that measured suicidal thoughts or behavior) was .18, which was only marginally different than 0 ($z = 1.80, p = .07$) and was only half as large as the average effect size of .34 for change in depressive symptoms across studies (Weisz et al. 2006). Given this chasm between treatment response in depression and suicidality, it is reasonable that factors associated with change in adolescent depressive symptoms, such as maternal depressive symptoms, may not necessarily translate to suicidal ideation.

Complicating this interpretation slightly is the finding that levels of adolescent depressive symptom and suicidal ideation were strongly correlated with each other at every time point (see Table 1), although we did not run a model with adolescent suicidal ideation *change* and

adolescent depression *change*. These correlated relationships are not surprising, given that suicidal ideation is an explicit criterion of MDD and is inherently linked to depression (e.g., Bridge et al. 2006). However, the change of the two phenomena did follow somewhat different trajectories and it is the trajectories that are the focus of the present study. Statistically, it is also possible that the lack of correlation between slopes of maternal depressive symptoms and adolescent suicidal ideation simply reflects the difficulty in detecting covariance of slopes in latent growth curve models, even in samples larger than this one (Hertzog et al. 2006).

Overall, study results are consistent with a family systems theory conceptualization and suggest that the mental health of each family member affects that of other individuals in the family (Hughes and Gullone 2008; Restifo and Bögels 2009). Though maternal depressive symptoms were not targeted in treatment, they declined concurrently with adolescent depressive symptoms. Given that adolescent depression can precipitate stressful events and circumstances within the family system (Rudolph et al. 2000), declines in adolescent depressive symptoms may have led to lower stress and conflict within the family, which, in turn, positively affected maternal mental health. Decreases in maternal depressive symptoms, in turn, may have led to a more positive parenting environment, characterized by greater warmth and less punitive parenting practices, as suggested by prior treatment research with depressed parents (Foster et al. 2008; Forehand et al. 2012). This positive change in family environment then may have led to further decreases in adolescent depressive symptoms and so on, particularly among youth with high levels of depressogenic inferential styles (Abela et al. 2006) or negative attachment cognitions (Abela et al. 2009), who tend to be more susceptible to the impact of parental depressive symptoms and behaviors.

Implications for Treatment

This study's results suggest that mothers with subclinical depressive symptoms experience positive changes in their own mental health when their adolescents receive treatment for depression. Thus, for mothers with subclinical depressive symptoms, standard treatment for adolescent depression that includes some parental involvement may be sufficient to incite change in both mothers and adolescents. It is not clear, however, whether this finding would remain true for mothers whose own depressive symptoms are more severe. This question deserves attention in future research. A more comprehensive and integrated treatment approach may be required for mothers with more severe depression, which optimally would include a full course of independent treatment for mothers (e.g., Foster et al. 2008). Under such conditions, an optimal protocol for parent-adolescent dyads who are *both* moderately to severely depressed might involve theoretically consistent concurrent treatment protocols for each member. This treatment likely would integrate family work to improve parent-child interactions, as well as individual sessions for parents and adolescents to work on skill building (including parenting skills) and openly discuss sensitive issues precipitating depressive symptoms.

The study's results also suggest the potential importance of regularly measuring family processes (e.g., parenting behaviors, cohesion, perceptions of support) and parental mental health symptoms as mechanisms of change in intervention research with depressed youth. In a meta-analysis, Weisz et al. (2006) found that most empirically-supported treatments measured cognitive processes (31 of 35), whereas far fewer (7 of 35) examined other mediators and/or moderators, and only three studies explored family processes (family functioning, family criticism and over-involvement) in relationship to outcomes (Diamond et al. 2002; Moldenhauer 2004; Rosello and Bernal 1999). For example, in one study, attachment-based family therapy led to a significantly greater reduction in depression,

anxiety and family conflict, in comparison to a waitlist group (Diamond et al. 2002). In a family-based cognitive behavioral depression prevention program, increases in positive parenting mediated intervention effects on adolescent internalizing and externalizing symptoms, accounting for approximately half of the effect on youth outcomes (Compas et al. 2010). Yet, in another study, neither CBT nor interpersonal therapy (IPT) led to a significant change in perceived parental criticism or intensity of familial emotional involvement in a sample of Puerto Rican adolescents, even though depressive symptoms did decline significantly (Rosello and Bernal 1999). Overall, family processes are understudied in relation to depression treatment outcomes and could benefit from further research (Weisz et al. 2006). Understanding these processes may help clarify the active ingredients in interventions with depressed youth and parents.

Limitations

This study has limitations that deserve mention. One potential limitation is the fact that mothers, on average, were not severely depressed. As mentioned, only 28 % of mothers reported BDI-II scores that denoted at least mild depression, and only 16 % reported BDI-II scores denoting moderate or higher-level symptoms. The relationship between change in maternal and adolescent symptoms may be more pronounced in parent-child dyads in which both members are more markedly depressed. Still, the vast majority of mothers (90 %) reported at least one symptom of depression. As sub-clinical depressive symptoms impair quality of life (e.g., Cuijpers et al. 2004; Howland et al. 2008), and predict future major depression (Fogel et al. 2006), research that examines the effects of interventions on subclinical depressive symptoms holds value. Indeed, a meta-analysis of treatment of subclinical depression suggests that treatment is effective and can prevent the onset of major depression (Cuijpers et al. 2007).

A second limitation is the reliance on self-report data for parental psychopathology and adolescent suicidal ideation. A diagnostic interview or clinician-rated data could provide more complete and objective measures of these variables. TORDIA, does however, use reliable and well-validated self-report measures that have been widely used in past studies, and thus these assessment tools can be considered sound. Third, this study did not include fathers or other guardians due to the small number of fathers who participated. Research to date has shown an association between paternal depression and offspring internalizing and externalizing symptoms (Kane and Garber 2004), though paternal factors may have a weaker relationship to child mental health than do maternal factors (e.g., Connell and Goodman 2002). This question would benefit from further investigation, especially as fathers' role in childcare continues to increase in many families (Hughes and Gullone 2008). Fourth, though not uncommon in studies with follow-up assessments that span multiple years (e.g., Curry et al. 2011), this study had a fairly low retention rate at later follow-up time points. By 72 weeks, 52 % of adolescents and 54 % of mothers who had completed baseline measures were retained. To minimize the effect of attrition, Full Information Maximum Likelihood (FIML) estimation was employed to account for missing data. FIML provides less-biased estimates than does listwise deletion. Still, the participants who were available for assessment at 72 weeks were, on average, less severely depressed and more likely to have improved during treatment than those who were not retained (Vitiello et al. 2011). Notably, these are not uncommon differences between adolescents who are retained and those who are not retained in large-scale longitudinal treatment studies (May et al. 2007). Moreover, a previous report (Vitiello et al. 2011) describes convergence between analyses with imputed values and those with last observation carried forward, suggesting that retention bias may not be a primary factor in explaining findings. Fifth, these analyses do not allow for examination of directionality, but do provide a good description of the trajectory of change in each group over time. Finally, the racial composition was somewhat

homogenous; future research that includes a larger number of minority participants is needed. As this is the first study to examine concurrent trajectories of change in parent and adolescent depressive symptoms in the context of a youth clinical trial, results are important, despite inherent limitations.

Future Directions

This study adds to the nascent body of literature supporting the longitudinal relationships between parent and child depression in community-based samples (e.g., Elgar et al. 2003; Forehand and McCombs 1988; Ge et al. 1995; Kouros and Garber 2010). Interestingly, these results also contradict previous research suggesting that maternal depressive symptoms relate to initial levels of adolescent depressive symptoms, but not to the course of these symptoms over time (e.g., Garber et al. 2002). In the present study, symptom trajectories were indeed correlated over the course of time, suggesting concurrent patterns of change. Study results also replicate findings from the youth anxiety literature, which suggest that reductions in children's anxiety post-treatment appear to influence later parental anxiety (Silverman et al. 2009). Notably, mothers in the present study were not severely depressed on average. Thus these results also set the stage for future research exploring the trajectories of symptom change in samples of adolescents and parents in which both members have clinical levels of depression.

It also would be of interest to extend the present research to examine the growth trajectories of other family-related factors in a clinical sample over the course of treatment. Promising constructs include those that have been shown to have a reciprocal effect on parent and adolescent functioning in community-based research, such as parental rejection (Hale et al. 2008), attachment to parents (Buist et al. 2004), and parental warmth (Doyle and Markiewicz 2005). Moderation analyses also could help clarify which family processes are most influential for which populations or conditions (e.g., gender, family beliefs about mental illness, family structure). Answers to such questions could inform the development, evaluation and dissemination of the next generation of interventions for adolescent mental health symptoms. Whenever possible, future researchers also should collect more data from fathers to expand our understanding of family dynamics beyond that of mother-child.

Conclusion

Prior research suggests that maternal depressive symptoms influence the onset of adolescent depressive symptoms (Garber et al. 2002) and the development of broader internalizing symptoms in youth (Fatori et al. 2013; Leve et al. 2005), but has not explored the inter-relationship between adolescent and maternal symptoms over the course of time. The present study used bivariate latent growth curve analysis to examine whether maternal and adolescent depressive symptoms fluctuate concurrently over the course of 72 weeks in the context of an adolescent depression treatment study. Results expand upon existing knowledge and suggest that adolescent and maternal depressive symptoms have correlated trajectories of decline. In other words, mothers' own mental health improves when their adolescents' depressive symptoms decline in treatment, even if the treatment does not directly target maternal symptoms. This relationship was most pronounced when analyses were limited to dyads that included mothers who reported depressive symptoms at the start of their adolescents' depression treatment. Thus, these results invite future research that examines whether similar patterns would be found in parent-adolescent dyads that include more severely depressed mothers. Attention to parent-adolescent trajectories of change in mental health symptoms over the course of treatment, and to family processes such as attachment, warmth or communication, may improve our understanding of mechanisms of change and ultimately lead to more tailored and effective interventions.

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Fig. 1.

Latent growth model of maternal depression and adolescent depression. A simplified depiction of the structural equation model used to estimate latent basis growth curves of maternal depression (*MD*) via BDI-II and adolescent depression (*AD*) via CDRS-R. The intercept path was constrained to 1 in all cases. The slope of each variable was constrained to 0 at 0 weeks and 72 at 72 weeks. Interim time points were estimated. Though not depicted, analyses controlled for gender and CBT condition on the latent intercepts and slopes. The error structure is not shown

Fig. 2.

Trajectories of growth over time in maternal depression, adolescent depression and adolescent suicidal ideation. *Clockwise from top-left*, the trajectories of decline over 72 weeks in adolescent depressive symptoms, adolescent suicidal ideation and maternal depressive symptoms. For all three variables, the rate of decline was steepest during the initial 12 weeks of treatment. Adolescent depression and suicidal ideation, which were also measured after 6 weeks, showed even faster decline during these initial 6 weeks

Table 1
Descriptive statistics and correlations among maternal depression, adolescent depression and adolescent suicidal ideation

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
<i>n</i>	241	214	185	149	131	333	292	287	259	196	173	328	289	280	249	189	168
<i>M</i>	10.43	7.43	7.91	5.90	5.80	58.82	42.46	37.47	33.04	31.60	29.92	41.62	34.13	31.53	28.23	27.96	25.15
<i>SD</i>	9.760	7.90	9.16	6.19	7.24	10.42	13.64	13.36	12.94	13.72	13.68	22.30	21.09	18.83	15.84	16.42	13.27
Maternal depression (BDI)																	
1. MD 0	–	.707**	.636**	.423**	.618**	.078	.006	.040	.077	–.038	–.085	.060	.037	.084	.113	.113	.117
2. MD 12		–	.771**	.491**	.638**	.039	.169*	.152*	.100	.090	–.171	–.037	–.030	.058	–.038	.069	–.009
3. MD 24			–	.640**	.666*	.075	.173*	.117	.207**	.162	–.102	–.052	.031	–.002	.019	.052	–.044
4. MD 48				–	.588**	.049	.049	.038	.181*	.317**	.056	.090	.092	.119	.114	.211*	.121
5. MD 72					–	–.053	–.021	.030	.017	.067	.052	–.081	–.121	–.078	–.061	–.035	–.094
Adolescent depression (CDRS-R)																	
6. AD 0						–	.373**	.424**	.286**	.405**	.243**	.369**	.223**	.266**	.259**	.327**	.189*
7. AD 6							–	.590**	.445**	.413**	.378**	.296**	.510**	.356**	.339**	.345**	.267**
8. AD 12								–	.580**	.446**	.462**	.243**	.365**	.491**	.444**	.287**	.330**
9. AD 24									–	.499**	.462**	.223**	.302**	.356**	.549**	.345**	.432**
10. AD 48										–	.692**	.217**	.294**	.290**	.437**	.551**	.457**
11. AD 72											–	.133	.225**	.290**	.308**	.357**	.473**
Adolescent suicidal ideation (SIQ)																	
12. AS 0												–	.675**	.651**	.596**	.488**	.439**
13. AS 6													–	.773**	.718**	.582**	.454**
14. AS 12														–	.754**	.601**	.565**
15. AS 24															–	.633**	.747**
16. AS 48																–	.720**
17. AS 72																	–

MD maternal depression, AD adolescent depression, AS adolescent suicidal ideation

* $p < .05$;

 $p < .01$

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Table 2

Modeled intercepts and slopes for maternal depressive symptoms and adolescent depressive symptoms

	Estimate (standard error)	Critical ratio	<i>p</i>
Adolescent depressive symptoms intercept	59.386 (.881)	67.378	***
Adolescent depressive symptoms slope	−399 (.019)	−21.210	***
Maternal depressive symptoms intercept	9.924 (.936)	10.607	***
Maternal depressive symptoms slope	−.060 (.012)	−4.880	***

 $p < .001$

Table 3

Modeled intercepts and slopes for maternal depressive symptoms and adolescent suicidal ideation

	Estimate (standard error)	Critical ratio	<i>p</i>
Adolescent suicidal ideation intercept	40.875 (1.993)	20.509	***
Adolescent suicidal ideation slope	-.231 (.027)	-8.611	***
Maternal depressive symptoms intercept	9.958 (.942)	10.570	***
Maternal depressive symptoms slope	-.062 (.013)	-4.850	***

 $p < .001$

Table 4

Structural correlations between intercepts and slopes of maternal depressive symptoms and adolescent depressive symptoms

	Estimate (standard error)	Critical ratio	<i>r</i>	<i>p</i>
Maternal depression intercept to maternal depression slope	–.355 (.124)	–2.867	–.732	**
Maternal depression slope to adolescent depression slope	.005 (.002)	2.127	.578	*
Maternal depression intercept to adolescent depression intercept	14.949 (8.208)	1.821	.214	.069
Adolescent depression intercept to adolescent depression slope	–.396 (.269)	–1.475	–.341	.140
Maternal depression intercept to adolescent depression slope	–.162 (.148)	–1.095	–.142	.274
Adolescent depression intercept to maternal depression slope	–.192 (.125)	–1.540	–.388	.123

Bold denotes a correlation that comprised a primary research question

**
p < .01;

*
p < .05

Table 5

Structural correlations between levels and slopes of maternal depressive symptoms and adolescent suicidal ideation (SI)

	Estimate (standard error)	Critical ratio	<i>r</i>	<i>p</i>
Maternal depression intercept to maternal depression slope	-.441 (.138)	-3.192	-.723	**
Maternal depression slope to adolescent SI slope	.001 (.003)	.289	.050	.772
Maternal depression intercept to adolescent SI intercept	14.622 (14.008)	1.044	.084	.297
Adolescent SI intercept to adolescent SI slope	-3.384 (.534)	-6.335	-.765	***
Maternal depression intercept to adolescent SI slope	.039 (.192)	.202	.021	.840
Adolescent SI intercept to maternal depression slope	-.316 (.192)	-1.642	-.218	.101

Bold denotes a correlation that comprised a primary research question

**
 $p < .01$;

 $p < .001$

Table 6

Parameter estimates for latent growth models

Weeks since baseline	<u>Latent basis estimate (standard error)</u>		
	Maternal depression	Adolescent depression	Adolescent suicidal ideation
0	.000	.000	.000
6	–	41.23 *** (1.89)	20.80 *** (3.58)
12	42.06 *** (5.44)	53.52 *** (1.93)	33.19 *** (3.01)
24	40.39 *** (6.37)	63.73 *** (2.14)	52.35 *** (2.62)
48	62.27 *** (6.32)	67.19 *** (2.35)	59.31 *** (3.48)
72	72.00	72.00	72.00

Weeks 0 and 72 were not estimated, but were set by the authors

 $p < .001$