ABSTRACT

Title of Thesis: RECURRENCE AND TIMING OF EXPOSURE TO MATERNAL DEPRESSION AND THE DEVELOPMENT OF DEPRESSIVE AND CONDUCT SYMPTOMS IN CHILDREN WITH AND WITHOUT ADHD

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The current study examined the longitudinal associations between recurrence and timing of exposure to maternal depression and the development of depressive and conduct symptoms in children with and without ADHD. Methods: 125 children with ADHD and 122 comparison children, ages 4-6, were followed over an 8-year period (until age 12-14). Results: Total recurrence of maternal depression was associated with youth depressive and conduct symptoms. Moreover, early adolescent exposure to maternal depression predicted youth depressive symptoms for all children. Exposure to maternal depression during preschool, childhood, and early adolescence each independently predicted youth conduct symptoms. Child ADHD status moderated the link between total recurrence of maternal depression and youth depressive symptoms and the link between preschool exposure and youth depressive symptoms. Child ADHD status did not moderate relations between total recurrence

and timing of exposure to maternal depression and youth conduct symptoms.

RECURRENCE AND TIMING OF EXPOSURE TO MATERNAL DEPRESSION AND THE DEVELOPMENT OF DEPRESSIVE AND CONDUCT SYMPTOMS IN CHILDREN WITH AND WITHOUT ADHD

by

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Dedication

To my parents.

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Chapter 1: Introduction

Compared to typically developing children, children with attention deficit/hyperactivity disorder (ADHD) are at increased risk for developing depression and conduct disorder (CD), particularly as they enter adolescence (Elia, Ambrosini, & Berrettini, 2008). Yoshimasu et al. (2012) reported that 62% of children (N=379) with childhood ADHD developed comorbid disorders by late adolescence. Comorbidity among children with ADHD predicts adverse developmental trajectories. For example, compared to youth with only depression or ADHD, children with comorbid ADHD and depression experienced greater social impairment (Blackman, Ostrander, & Herman, 2005), increased rates of other psychiatric disorders (Biederman, Faraone, Milberger, & Guite, 1996), and higher rates of psychiatric hospitalizations (Biederman et al., 2008; Biederman et al., 1996). Compared to youth with ADHD and/or oppositional defiant disorder (ODD), children with ADHD and CD exhibited more severe delinquent behaviors at earlier ages (Sibley et al., 2011), increased substance use disorders (Lee, Humphreys, Flory, Liu, & Glass, 2011) and higher rates of delinquency into adulthood (Mordre, Groholt, Kjelsberg, Sandstad, & Myhre, 2011). Lastly, comorbid CD and depression among children with ADHD predicted two of the most devastating and costly mental health outcomes—*criminality* (Satterfield et al., 2007) and *suicide* (James, Lai, & Dahl, 2004), respectively.

In addition to being at greater risk for meeting diagnostic criteria for depression and CD, youth with ADHD also experienced higher rates of continuous depressive and conduct symptoms than comparison children (Lahey et al., 2007). From a developmental psychopathology perspective, the onset of ADHD typically occurs long before the steep emergence of both depressive and conduct disorders in adolescence (Costello, Mustillo, Erkanli, Keeler & Angold, 2003). Thus, identifying precursors to the development of depressive and conduct symptoms in

young children with ADHD has great potential to target children who may benefit most from early intervention and prevention efforts.

Maternal Depression and the Course of Adult Depression

Maternal depression is a robust predictor of depression and CD among children with ADHD. In a sample of children with ADHD, children of mothers with a lifetime history of major depressive disorder (MDD) exhibited higher rates of conduct problems over an eight-year period compared to children of never depressed mothers (Chronis et al., 2007). Additionally, among children with and without ADHD, lifetime maternal depression increased risk for youth MDD by age 18 two-fold (Chronis-Tuscano et al., 2010).

However, the aforementioned studies have been based on crude measurements of maternal depression, typically assessed via a dichotomous, "ever-or-never" variable measured at a single time point. Yet, adult depression is a heterogeneous disorder, varying in many facets. Specifically, recurrence of depression is common and the rates of recurrence increase with every subsequent episode (Mueller et al., 1999). Studies that examined the recurrence of adult depression after 15 years have reported rates ranging from 35% to 85% (Eaton et al., 2008; Mueller et al., 1999). Therefore, given the heterogeneity in the course of adult depression, a more nuanced examination of the relation between the course of maternal depression and child emotional and behavioral outcomes is warranted.

Recurrence of Maternal Depression

Because adult depression is often recurrent (Eaton et al., 2008; Mueller et al., 1999), children may be exposed to various levels of recurrence to maternal depression. Studies indicate recurrent maternal depression predicts youth internalizing and externalizing outcomes. For example, compared to offspring of never depressed mothers, offspring of mothers who experienced chronic levels of depression have higher rates of: adolescent self-reported

depression, internalizing, and externalizing problems (Campbell, Morgan-Lopez, Cox, & McLoyd, 2009); teacher-reported externalizing-aggressive behaviors and parent-reported externalizing-ADHD behaviors (Ashman, Dawson, & Panagiotides, 2008) and adolescent-reported suicidal ideation (Hammerton et al., 2015). Additionally, the effects of maternal antenatal (i.e. depression during pregnancy) and post-partum depression on subsequent offspring depression have received wide attention in the literature (Goodman, 2007). However, studies suggest these relations may be better explained by recurrence to maternal depression (Pawlby, Hay, Sharp, Waters, & O'Keane, 2009; Raposa, Hammnen, Brennan & Najman, 2014; Sanger et al. 2015). While the literature on recurrence to maternal depression is replete, studies examining total recurrence continuously during a wider range of time or examining outcomes into early adolescence are still needed.

Developmental Considerations for Children with ADHD

Given the recurrent nature of adult depression and associated outcomes with youth emotional and behavioral functioning, children may be exposed to maternal depression during multiple developmental periods. To date, the relationship between the timing and recurrence of maternal depression and the emergence of subsequent depressive and conduct symptoms in offspring remains understudied. However, several developmental considerations suggest that timing and recurrence of maternal depression may have differential effects on child emotional and behavioral outcomes, and, perhaps, particularly for children with ADHD.

Exposure to maternal depression during the preschool period may disrupt the myriad of developmental processes occurring at that stage, such as optimal brain development (Giedd et al., 1996), attachment security (Bowlby, 1977), and language abilities (McCarthy, 1933). Children with ADHD in particular show delays in their emotional and biological maturity compared to same-age peers (Minde et al., 2003; Krain & Castellanos, 2006), and therefore may require

additional caregiver assistance. Because children rely more heavily on their caregivers during this stage, caregiver depression may disrupt or delay the attainment of necessary developmental skills, posing a continuous challenge to child functioning.

During childhood, children undergo a major transition into the school setting. Children with ADHD often experience more social and academic difficulty in structured, academic environments and require additional accommodations (Raggi & Chronis, 2006). Caregiver depression may disrupt the caregiver's ability to optimally facilitate these difficult transitions.

Lastly, early adolescence represents a developmental juncture characterized by an increase in risk-taking coupled with increases in the importance of autonomy (Steinberg & Silverberg, 1986) and peer relationships (La Greca & Harrison, 2005). Youth with ADHD struggle with behavioral inhibition and decision-making, and are more likely to gravitate toward deviant peers (Marshal, Molina, & Pelham, 2003). This tendency makes an already vulnerable period possibly more risky for youth with ADHD compared to that of same-age peers. Additionally, the steep emergence of both depression and conduct disorder occurs during early adolescence (Costello et al., 2003). Thus, exposure to maternal depression during early adolescence may decrease protective familial mechanisms that have been found to curtail the development of negative emotional and behavioral outcomes in youth (Brennan, Le Broque & Hammen, 2003).

Timing of Exposure to Maternal Depression

Given the often recurrent course of adult depression as well as the various milestones unique to each child developmental stage, studies have begun to shed light on the relation between timing of exposure to maternal depression and child internalizing and externalizing outcomes. While such studies have primarily been *limited* to examining exposure during early childhood (i.e., primarily up to child age 5; Ashman et al., 2008; Bagner, Pettit, Lewinsohn, &

Seeley, 2010; Brennan et al., 2000; Dougherty, Tolep, Smith, & Rose, 2013; Essex, Klein, Miech, & Smider, 2001; Kim-Cohen, Moffitt, Taylor, Pawlby, & Caspi, 2005; Barker, 2013), common findings are clear: the relation between maternal depression and child internalizing and externalizing outcomes appears to be contingent on the timing of exposure during early childhood.

Only a few studies have extended these findings beyond exposure to maternal depression during early childhood (Halligan, Murray, Martins, & Cooper, 2007; Hay, Pawlby, Waters, Perra & Sharp, 2010; Hay, Pawlby, Waters, & Sharp, 2008; Pearson et al. 2013; Gajos & Beaver, 2015). Some of these studies have found that late maternal depression (defined as cumulative exposure to maternal depression during child age 1-16) predicts youth emotional and disruptive behavior disorders above maternal antenatal and postpartum depression (Hay et al. 2008). Other studies have found antenatal and/or postpartum depression uniquely predicts child depressive outcomes at age 18 (Pearson et al., 2013) and antisocial behavior at age 16 (Hay et al. 2010) above that of late maternal depression (defined as exposure during child age 12 or between child age 4-11, respectively). Moreover, additional studies have found that both early and late maternal depression predicted elevated levels of youth depressive outcomes (Halligan et al. 2007) and antisocial behavior (Gajos & Beaver, 2015). Only one study has found that timing of maternal depression does not differentially predict child internalizing and externalizing outcomes (Hammen & Brennan, 2003). However, in this study timing of exposure was examined among mothers who had only experienced a single depressive episode, which is an atypical subpopulation.

Taken together, the aforementioned studies suggest late maternal depression predicted child depressive and conduct outcomes either in conjunction with, or above, early exposure to maternal depression. However, these studies are similarly limited in several aspects. A pervasive

issue is that the operationalization of "late" maternal depression often intertwines multiple child developmental periods, removing the opportunity to examine unique effects of exposure during more specific and meaningful stages (Halligen et al., 2007; Hay et al., 2008). Other definitional issues include operationalizing "late" maternal depression as a self-report of depression at one time point during a range of years (Pearson et al., 2013; Gajos & Beaver, 2015), not examining effects of concurrent maternal depression (Pearson et al., 2013; Hay et al., 2010), querying about the last several years, and thus rely on retrospective memory, in diagnostic interviews (Halligan et al., 2007; Hay et al., 2008; 2010), and examining outcomes within a range of child ages (Halligan et al., 2007; Hay et al., 2008). Lastly, arguably the largest issue is the lack of theoretical rationale for operationalizing "late" maternal depression in such manners. Thus, our current understanding of the effects of late maternal depression is limited by both theoretical and methodological shortcomings.

Summary of Gaps in Literature

Despite the strong comorbidity with depression and conduct problems among youth with ADHD (Elia et al., 2008), the documented evidence that maternal depression is a strong predictor of these comorbidities (Chronis et al., 2007; Chronis-Tuscano et al., 2010), and studies showing depression in adults is often recurrent (Richards, 2011), *no study* has examined the timing and recurrence of maternal depression as predictors of the development of depressive and conduct symptoms among children with and without ADHD during early adolescence, a highly vulnerable time period.

The Current Study

The current study addressed these gaps in the literature by examining the longitudinal association between recurrence and timing of maternal depression during multiple, meaningful developmental periods and the development of depressive and conduct symptoms in early

adolescence (at ages 12-14 years) among children with and without ADHD. Maternal depression was examined using a methodologically refined manner, as annual assessments with structured diagnostic interviews were used to examine maternal depression. Child depressive outcomes were assessed using diagnostic interviews from both youth and parent report; measurement of conduct outcomes additionally included teacher reports.

Aim 1

The first aim of the current study was to examine the longitudinal association between total recurrence of (i.e., total exposure to) maternal depression and the development of youth depressive and conduct symptoms during early adolescence.

Hypothesis 1: Increased recurrence of (i.e., total exposure to) maternal depression would predict the development of youth depressive and conduct symptoms during early adolescence.

Given that depression is a chronic condition (Eaton et al., 2008; Mueller et al., 1999) and that recurrent maternal depression predicts youth internalizing and externalizing outcomes (e.g., Ashman et al., 2008; Campbell et al., 2009; Luoma et al., 2001; Pawlby et al., 2009), it was hypothesized that increased recurrence of maternal depression would predict higher rates of depressive and conduct symptoms for all youth.

Aim 2

The second aim was to examine the association between timing of exposure to maternal depression and the development of youth depressive and conduct symptoms during early adolescence.

Hypothesis 2: Differential timing of exposure to maternal depression would predict the development of depressive and conduct symptoms during early adolescence.

On one hand, exposure to maternal depression during preschool may disrupt the

development of fundamental socio-emotional, cognitive, and language skills, posing a continuous challenge to child functioning in later development. This is the theoretical perspective most of the literature has taken, as many studies examining timing of maternal depression have focused on early childhood exposure (Ashman et al., 2008; Bagner et al., 2010; Brennan et al., 2000; Dougherty et al., 2013; Essex et al., 2001; Kim-Cohen et al., 2005; Barker, 2013).

The literature on timing of maternal depression across developmental periods aside from the early childhood period is limited. And of the studies examining "late" maternal depression, some studies found late maternal depression predicted child internalizing and externalizing outcomes above early maternal depression (Hay et al., 2008). In other cases, studies found late and early maternal depression jointly predicted child outcomes (Halligan et al., 2007; Gajos & Beaver, 2015). In yet other studies, early maternal depression predicted child outcomes above late maternal depression (Pearson et al., 2013; Hay et al, 2010). No prior study, to our knowledge, has examined the relationship between timing of exposure to maternal depression and child emotional and behavioral outcomes in a methodologically refined manner. Thus, we do not have specific hypotheses regarding which exposure period will be most predictive of child outcomes.

Aim 3

The third aim was to examine whether child ADHD status moderates the relationship between recurrence and timing of maternal depression and youth depressive and conduct outcomes during early adolescence.

Hypothesis 3: Child ADHD status would moderate the relationship between total recurrence of maternal depression and the development of youth depressive and conduct symptoms during early adolescence. The association between recurrence of maternal

depression and youth depressive and conduct outcomes would be stronger in children with ADHD relative to that of children in the comparison group.

Approximately 40% of children with ADHD have mothers with histories of depression (Chronis et al., 2003), and maternal depression is a robust predictor of both depression and conduct disorder in children with ADHD (Chronis et al. 2007; Chronis-Tuscano et al., 2010). Children with ADHD are more likely to require continuous parental assistance with mastering developmental tasks compared to same age peers. Thus, recurrent maternal depression may be a stronger predictor of both depressive and conduct symptoms in children with ADHD relative to comparison children, as recurrent depression may further impair parental assistance.

Hypothesis 4: Child ADHD status would moderate the relations among timing of exposure to maternal depression and the development of youth depressive and conduct symptoms during early adolescence. These relations are hypothesized to be stronger among children with ADHD relative to that of comparison children.

Children with ADHD encounter unique developmental challenges during preschool, childhood, and early adolescence. Delays in emotional and biological maturity, social and academic impairments, and gravitation toward deviant peers afflict children with ADHD across development. Therefore, timing of exposure to maternal depression may predict higher rates of depressive and conduct symptoms among children with ADHD, as exposure might further disrupt developmental processes during each unique period.

Chapter 2: Methods

Participants

Participants included 4-6 year old children with and without ADHD (N=247; 125 ADHD, 122 Comparison) and their biological mothers. Participants were recruited in two cohorts from clinical care settings in Chicago, Illinois and Pittsburgh, Pennsylvania. In Pittsburgh, about half of the participants were also recruited through community advertisements. Participants with ADHD did not differ based on recruitment source (Lahey et al., 1998).

Comparison participants were recruited to match participants with ADHD on demographic variables, such as sex, race/ethnic background, and age. Comparison participants were recruited from similar schools and neighborhoods. Exclusionary criteria for comparison participants included having met diagnostic criteria for ADHD and having been referred for any mental health problems in the past. However, comparison participants were not excluded if they met criteria for a disorder other than ADHD at baseline.

In addition, inclusionary criteria for all participants included enrollment in a structured educational setting (i.e. school) and co-residence with their biological mothers. Children were excluded from the study during the screening process if they met criteria for pervasive developmental disorder, mental retardation, or seizure disorder (5 participants were excluded for these reasons). Additionally, four children with intelligence scores < 70 based on the Stanford-Binet Intelligence Test – Short version (Thorndike, Hagen, & Sattler, 1986) were later excluded (Lahey et al., 2004). In total, three hundred and ten participants were eligible, and 259 participated. The current sample (n=247) represents 95.3% of the total sample and includes only participants who had a group identified (i.e. ADHD or comparison). Demographic information

for all participants at baseline is presented in Table 1.

Procedures

Mothers and children were consented in clinics in Pittsburgh and Chicago and were interviewed by two masked interviewers. Interviewers had at least a bachelor's degree in psychology, social work, or education and had previous experience working with children. All interviewers underwent extensive training. Both groups of participants were assessed each year for eight years (except for year 5, where there was a lapse in funding). If families failed to participate in one follow-up year, they typically participated in the next follow-up year. At year 2, 3, 4, 6, 7, 8, 9, at least some data were present for 91%, 90%, 90%, 90%, 87%, 88%, and 88% of participants, respectively. At the outcome assessment (year 9), both parent and youth assessments were obtained 94% of the time and teacher ratings of youth conduct symptoms were available for 70% of participants.

Measures

Child ADHD Diagnoses – At baseline, to meet *symptom criteria* for ADHD based on DSM-IV criteria, each ADHD symptom was considered present if endorsed by either parent or teacher (Shemmassian & Lee, 2015). Parent report of symptoms was based on responses to the Diagnostic Interview Schedule for Children (DISC; Shaffer, Schwab-Stone, Fisher, & Cohen, 1993). Teacher report of symptoms was based on the Disruptive Behavior Disorders Rating Scale (DBDRS; Pelham, Gnagy, Greenslade, & Milich, 1992; See Appendix A), which was sent to teachers by mail. If teacher reported any symptom as being "pretty much" or "very much" present, this was considered positive endorsement of that symptom. Children met ADHD symptom criteria by having 6 out of 9 symptoms for hyperactivity and/or 6 out of 9 symptoms for inattention based on parent or teacher-report.

Then, based on DSM-IV *diagnostic criteria* for ADHD, impairment was assessed in two

separate ways. First, using the DISC, parents were queried about whether the child's ADHD symptoms had caused problems at home, school, or with peers. Parent report of problems in any of these settings was considered impairing. Second, parents and teachers completed the Impairment Rating Scale (IRS; Fabiano et al., 2006; See Appendix B and C), which assesses impairment through children's need for treatment across multiple situations (e.g., at home, with peers). To meet criteria for impairment on the IRS, the parents or teacher had to rate the child at least a "3" (scale 0-6) in at least one area (e.g. school, home, and/or peers)[1].

Final ADHD diagnoses at baseline were made if: (1) child met ADHD symptom criteria (6 out of 9 symptoms on hyperactivity and/or inattentive subtype scales); and (2) child met impairment criteria in *at least* one setting (home and/or school). In total, 125 children were classified in the ADHD group and 122 children were classified in the comparison group based on these criteria.

Maternal Depression – The Structured Clinical Interview for DSM-III-R, Non-Patient Edition (SCID-NP; Spitzer, Williams, Gibbon, & First, 1990; See Appendix D) was used to assess for depression in biological maternal caregivers. The SCID-NP is a widely used semi-structured clinical interview with adequate psychometric properties and assesses for lifetime and current DSM-III-R disorders. The major depressive disorder module was used to assess depression in biological mothers in years 1-9. At baseline, both current and lifetime maternal depression were assessed. During subsequent, yearly follow-up assessments, maternal depressive episodes in the last 12 months were assessed. Depression diagnoses were made based on DSM-III criteria, and diagnoses were dummy coded (absent/present) for each year.

[1] Children were not required to meet DSM-IV cross-situational impairment due to the young age (4-6 years old) of the children at year 1, consistent with other work from this longitudinal study (Chronis-Tuscano et al., 2010; Lahey et al., 2011). At this age, children with ADHD may not exhibit much impairment in school, given limited academic demands. During follow-up assessments, 96% of the children who were impaired in at least one setting initially met cross-situational impairment (home/peer and school) at a subsequent wave.

Total recurrence of maternal depression was operationalized as total number of waves in which the maternal caregiver met DSM-III-R criteria for depression during year 1-9 (range 0-8).

Because each assessment wave overlapped with multiple developmental periods, data were recoded from year to child age (e.g. year 1, baseline, was recoded into child age 4, 5, 6). Thus, *timing of exposure to maternal depression* was operationalized using the following: *Preschool Exposure* was operationalized as exposure to maternal depression anytime between child age 4-6*; Childhood Exposure* was operationalized as exposure to maternal depression anytime between child age 7-10*; Early Adolescent Exposure* was operationalized as exposure to maternal depression anytime between child age 11-14. All three exposure variables were dummy coded (absent/present). Developmental theory regarding the tasks children are attempting to master at each of these periods as well as the unique challenges children with ADHD face were used to inform the classification of these three periods.

Youth Depressive and Conduct Symptoms at Age 12-14 – To assess for youth depressive and conduct outcomes continuously, the DISC (Shaffer et al., 1993) was separately administered to the maternal caregiver and youth themselves. The DISC was administered based on the last 12 months at child age 12, 13 or 14, depending on child age at enrollment (e.g., children enrolled at age 4 were age 12 at the outcome assessment). In addition, for youth conduct outcomes only, the teacher also completed the DBDRS about the youth in the last 12 months at the outcome assessment (age 12, 13 or 14). Depressive and conduct outcomes were defined as the total number of symptoms endorsed by each informant separately.

Chapter 3: Data Analytic Strategy

We used Little's Missing Completely at Random (MCAR) test (Little, 1988) in SPSS v. 22 to examine patterns of missing data. Little's MCAR test indicated data were missing completely at random (X^2 (df=70)=74.04, p=.348). Subsequent analyses were conducted using *Mplus 7.1* (Muthen and Muthen, 1998-2012). We used multiple imputation using Bayesian analysis to handle missing data, which is superior to traditional methods, such as handling missing data with mean value or listwise deletion (Schafer & Graham, 2002). Analyses were performed using 100 imputed datasets (Graham et al. 2007).

In order to examine the hypothesized relations between total recurrence and timing of exposure to maternal depression and youth depressive and conduct symptoms during early adolescence, we tested a series of structural equation models (SEM). Four fit indices were used to estimate model fit: the χ^2 statistic, the Comparative Fit Index (CFI; Bentler 1990), the Standardized Root Mean Square Residual (SRMR; Hu & Bentler, 1999) and the Root Mean Square Error of Approximation (RMSEA; Steiger 1990). While nonsignificant χ^2 values indicate good fit, this index is sensitive to sample size (Kline, 2005). CFI values of .90 or higher are considered acceptable (Bentler, 1989). RMSEA values less than .05 are considered indicative of good fit, values between .05 and .08 indicate fair fit, and values greater than .10 indicate poor fit (Browne, Cudeck, & Bollen, 1993). SRMR values less than .08 indicate good fit (Hu & Bentler, 1999).

Youth depressive symptoms were operationalized as a latent variable created based on parent and youth report of youth depressive symptoms on the DISC at youth age 12-14. Parent and youth reports of youth depressive symptoms were significantly correlated (r=.471, p<.001), suggesting a latent variable measurement approach was appropriate. Similarly, youth conduct

symptoms were operationalized as a latent variable created based on parent and youth report of youth conduct symptoms on the DISC and teacher report of youth conduct symptoms on the DBDRS at youth age 12-14. Parent and teacher report (r=.225, p<.001), parent and youth report (r=.329, p<.001) and youth and teacher report (r=.467, p<.001) of youth conduct symptoms were significantly correlated, suggesting that a latent variable measurement approach was appropriate.

All subsequent analyses reported below were conducted controlling for child race, maternal education, family income, child sex, child intelligence, and child ADHD status (see Table 1 for descriptive information). Covariates were chosen based on their theoretical relationship with maternal depression and youth depressive and conduct outcomes[2].

[2] Results were also conducted using only significant covariates in the model. Results remained the same and the model fit was generally worse across the majority of analyses

Chapter 4: Results

Preliminary Analyses

Table 1 includes sample characteristics and descriptive statistics. Table 2 presents the correlations among all variables. Descriptive statistics indicated that, at baseline, 38.4% of mothers of children with ADHD and 23.8% of mothers of comparison children met criteria for lifetime depression. This is in line with prior rates of depression in caregivers among children with ADHD reported in the literature (Faraone et al., 2000). Total recurrence of maternal depression and all three exposure variables (preschool, childhood, and early adolescence) were correlated with youth depressive symptoms during early adolescence. Results were similar for youth conduct symptoms, with the exception that preschool exposure to maternal depression did not correlate with youth conduct outcomes (see Table 2).

Predicting Youth Depressive and Conduct Symptoms at age 12-14

Aim 1

Hypothesis 1: To test whether total recurrence of maternal depression (defined as total years depressed) predicted youth depressive and conduct symptoms during early adolescence (child age 12-14), total recurrence and all covariates (i.e. child race, maternal education, family income, child sex, child intelligence, and child ADHD status) were entered into the model. See Table 3 for a summary of model results.

Depressive outcomes – The model predicting youth depressive symptoms demonstrated good fit for the data, RMSEA=.044, CFI=.968, SRMR=.025, $X^2(df = 8) = 11.767$, p=.162. Total recurrence of maternal depression was a significant predictor of youth depressive symptoms during early adolescence (B=.469, $p < .001$), such that higher recurrence of maternal depression predicted higher youth depressive symptoms in this model.

Conduct outcomes – The model predicting youth conduct symptoms demonstrated good fit for the data, RMSEA=.025, CFI=.975, SRMR=.027, $X^2(df=18) = 20.714$, p=.294). Total recurrence of maternal depression was a significant predictor of youth conduct symptoms during early adolescence (B=.272, $p < .001$), such that higher recurrence predicted higher youth conduct symptoms in this model.

Aim 2

Hypothesis 2: To test the unique effects of preschool, childhood, and early adolescent exposure to maternal depression on youth depressive and conduct symptoms during early adolescence (child age 12-14), the three dummy-coded exposure variables, (i.e. exposure during preschool, childhood, and early adolescence) were entered into the models simultaneously along with all covariates (i.e., child race, maternal education, family income, child sex, child intelligence, and child ADHD status). See Table 3 for a summary of model results.

Depressive symptoms – The model predicting youth depressive symptoms demonstrated good fit for the data, RMSEA=.041, CFI=.965, SRMR=.025, X^2 (df=10)=14.112, p =.168. When all three exposure variables were simultaneously entered into the model, results demonstrated that preschool (B=.029, p=.706) and childhood (B=.020, p=.810) exposure to maternal depression were not significant predictors of youth depressive symptoms during early adolescence. However, early adolescent exposure to maternal depression was a significant predictor of youth depressive symptoms (B=.487, p<.001), such that early adolescent exposure was related to higher youth depressive symptoms in this model.

Conduct symptoms – The model predicting youth conduct symptoms demonstrated good fit for the data, RMSEA =.025, CFI =.973, SRMR =.026, X^2 (df=22)=35.399, p=.278. When all three exposure variables were simultaneously entered into the model, results demonstrated that preschool (B=-.178, p=.022), childhood (B=.218, p=.010), and early

adolescent (B=.310, p<.001) exposure to maternal depression each independently predicted youth conduct symptoms during early adolescence. Whereas childhood and early adolescence exposure predicted higher conduct symptoms, preschool exposure predicted lower conduct symptoms in this model.

Aim 3

Hypothesis 3: To examine whether child ADHD status moderated the relationship between total recurrence of maternal depression and youth depressive and conduct symptoms during early adolescence, the main effect of total recurrence and the interaction between child ADHD status and total recurrence were both entered into the model along with all covariates. See Table 3 for a summary of model results.

Depressive Symptoms – The model predicting youth depressive symptoms demonstrated good fit for the data, RMSEA=.037, CFI=.975, SRMR=.023, X^2 (df=9)=12.068, p=.210. There was a significant interaction between total recurrence and child ADHD status on youth depressive symptoms in this model (B=.290, p=.022). The interaction was interpreted using multi-group analysis. Total recurrence was a significant predictor of depressive symptoms in both groups; however, this relation was stronger among children with ADHD (ADHD: B=.615, p<.001; Comparison: B=.263, p=.033). The main effect of total recurrence on youth depressive symptoms during early adolescence remained significant in this model (B=.254, p=.035).

Conduct Symptoms – The model predicting youth conduct symptoms demonstrated good fit for the data, RMSEA=.016, CFI=.989, SRMR=.027, X^2(df=20)=21.187, p=.386. There was neither a main effect of total recurrence (B=.136, p=.277), nor an interaction between total recurrence and child ADHD status on youth conduct symptoms during early adolescence in this model (B=.186, p=.163).

Hypothesis 4: To test, in three separate models, whether child ADHD status moderates

the relation between exposure to maternal depression during each developmental period (preschool, childhood, early adolescence) and youth depressive and conduct symptoms during early adolescence. The main effects of preschool, childhood and early adolescent exposure to maternal depression as well as the interaction between child ADHD status and the relevant developmental period (e.g. preschool exposure) were entered as predictors along with all covariates. See Table 3 for a summary of model results.

Depressive Symptoms

Preschool Exposure X ADHD – The model predicting youth depressive symptoms demonstrated excellent fit for the data RMSEA=.032, CFI=.977, SRMR=.024, X^2 (*df*=11)= 13.714, *p*=.250. There was an interaction between preschool exposure to maternal depression and child ADHD status on youth depressive symptoms during early adolescence in this model (*B*=.255, *p*=.033). The interaction was interpreted using multi-group analysis. The relation between preschool exposure to maternal depression and youth depressive symptoms during early adolescence was only significant in children with ADHD (ADHD: *B*=.270, *p*=.022; Comparison: *B*=.052, *p*=.646). Additionally, the main effect of early adolescent exposure to maternal depression remained a significant predictor of youth depressive symptoms in this model (*B*=.488, *p*<.001)

Childhood Exposure X ADHD – The model predicting youth depressive symptoms demonstrated excellent fit for the data RMSEA=.035, CFI=.971, SRMR=.023, X^2 (*df*=11) =14.391, *p*=.212. The interaction between childhood exposure and child ADHD status was not a significant predictor of youth depressive symptoms (*B*=.128, *p*=.271). However, the main effect of early adolescent exposure remained a significant predictor of youth depressive symptoms in this model (*B*=.472, *p*<.001).

Early Adolescent Exposure X ADHD – The model predicting youth depressive

symptoms demonstrated good fit for the data RMSEA=.033, CFI=.972, SRMR=.024, X^2 (*df*=11) =13.916, *p*=.240. The interaction between child ADHD status and early adolescent exposure to maternal depression was not a significant predictor of youth depressive symptoms (*B*=.128, *p*=.291). The main effect of early adolescent exposure remained a significant predictor of youth depressive symptoms for all offspring in this model (*B*=.389, *p*<.001). Thus, early adolescent exposure to maternal depression was related to higher youth depressive symptoms for all children, regardless of ADHD diagnosis.

Conduct Symptoms

Preschool Exposure X ADHD – The model predicting youth conduct symptoms demonstrated excellent fit for the data, RMSEA=.026, CFI =.967, SRMR=.028, X^2(*df*=24) = 28.064, *p*=.257. The interaction between child ADHD status and preschool exposure to maternal depression was not a significant predictor of youth conduct symptoms during early adolescence in this model (*B*=.016, *p*=.901). The main effect of preschool exposure was not a significant predictor of youth conduct symptoms (*B*=-.190, *p*=.110) in this model; however, the main effects of childhood exposure (*B*=.218, *p*=.010) and early adolescent exposure (*B*=.311, *p*<.001) remained significant predictors of youth conduct symptoms.

Childhood Exposure X ADHD – The model predicting youth conduct symptoms demonstrated excellent fit for the data, RMSEA=.018, CFI=.984, SRMR=.025, X^2 (*df*=24)=25.992, *p*=.356. The interaction between child ADHD status and childhood exposure was not a significant predictor of youth conduct symptoms during early adolescence (*B*=.154, *p*=.195). However, whereas the main effect of childhood exposure was not a significant predictor of conduct symptoms (*B*=.112, *p*=.333), the main effect of preschool exposure (*B*=-.179, *p*=.021) and early adolescent exposure (*B*=.302 *p*<.001) remained significant predictors of youth conduct symptoms during early adolescence in this model.

Early Adolescence X ADHD – The model predicting youth conduct symptoms demonstrated excellent fit for the data, RMSEA=.024, CFI=.973, SRMR=.029, X^2 (*df*=24)=27.353, *p*=.288. The interaction between child ADHD status and early adolescent exposure was not a significant predictor of youth conduct symptoms during early adolescence (*B*=.052, *p*=.401). However, the main effects of preschool exposure (*B*=-.176, *p*=.024), childhood exposure (*B*=.217, *p*=.010), and early adolescent exposure (*B*=.275, *p*=.019) remained significant predictors of youth conduct symptoms for all children, regardless of ADHD diagnosis in this model. Whereas preschool exposure to maternal depression predicted fewer conduct symptoms, childhood exposure and early adolescent exposure predicted more conduct symptoms.

Chapter 5: Discussion

The current study extended the literature by examining timing and recurrence of maternal depression as predictors of adverse outcomes among children with and without ADHD. Results indicated that total recurrence of maternal depression and exposure to maternal depression during early adolescence predicted the development of depressive symptoms during early adolescence for all children. Also, across the entire sample, total recurrence of maternal depression, childhood exposure, and early adolescent exposure predicted more youth conduct symptoms. Child ADHD did not moderate associations between maternal depression and youth conduct symptoms. However, total recurrence of maternal depression and preschool exposure increased risk for youth depressive outcomes among children with ADHD.

Results demonstrated that total recurrence of maternal depression predicted youth depressive and conduct symptoms for all youth. This finding is in line with the literature demonstrating that children of mothers with recurrent depression experience more internalizing and externalizing problems across development (Ashman et al., 2008; Campbell et al., 2009; Pawlby et al., 2009). Study results replicated and extended knowledge in this area by demonstrating that recurrence of maternal depression measured annually across eight years predicted child functioning into early adolescence.

Results also demonstrated that early adolescent exposure to maternal depression, but neither preschool nor childhood exposure, uniquely predicted higher youth depressive symptoms for all youth. This finding uniquely adds to the literature, as most studies primarily focus on timing of maternal depression experienced during early childhood (Ashman et al., 2008; Bagner et al., 2010; Brennan et al., 2000; Dougherty et al., 2013; Essex et al., 2001; Kim-Cohen et al., 2005; Barker, 2013). Our findings indicated that early adolescent exposure to maternal

depression uniquely predicted youth depressive symptoms, even when controlling for exposure during preschool and childhood. Such findings are in line with findings by Hay et al. (2008) and Halligan et al. (2007). However, our study findings conflict with that of Pawlby et al. (2009) and Pearson et al. (2013). Such disparities could be due to different operational definitions of late maternal depression. For example, Pawlby et al. (2009) operationalized timing effects as child's first ever exposure to maternal depression, rather than the occurrence of any exposure during a specific period. Pearson et al. (2013) operationalized late maternal depression as depression at one time point (child age 12). Thus, different operationalizations of late maternal depression likely contributed to discrepant results.

Hammen's (1991) intergenerational interpersonal stress model of depression claims the association between maternal and youth depression is due to shared, stressful interpersonal situations, which then affects youth social functioning. Deleterious effects of early exposure (i.e. preschool or childhood) to maternal depression may be attenuated by later protective factors that emerge in importance during early adolescence (i.e., academic achievement or positive peer relations). At the same time, common stressors during early adolescence (e.g. parent-child conflict) that are linked to current exposure to maternal depression during early adolescence may exacerbate youth psychosocial functioning. Alternatively, exposure to maternal depression during early adolescence also coincides with the steep emergence in youth depressive symptoms, which may also contribute to study findings. One caveat to our finding is that early adolescent exposure was defined in our study as exposure to maternal depression between child age 11-14, and youth outcomes were examined at age 12-14. Thus, it could be that exposure during early adolescence was a stronger predictor of youth depressive symptoms because it was measured most proximally to the outcome assessment (Mars et al., 2012).

Our study findings also indicated that childhood and early adolescent exposure to

maternal depression predicted higher child conduct symptoms during early adolescence for all youth. Results are in line with those reported by Gajos and Beaver (2015) and Hay et al. (2008), who found that late maternal depression predicted youth antisocial behavior and disruptive behavior disorders either in conjunction with or above early exposure to maternal depression. However, findings conflict with that of Hay et al. (2010), who reported maternal antenatal depression (i.e. depression during pregnancy) predicted youth antisocial behavior at age 16 above exposure to maternal depression during later developmental periods. One explanation for the disparity is Hay et al. (2010) did not examine concurrent maternal depression. Hay et al. (2010) also dichotomously defined antisocial behavior as having met diagnostic threshold for CD and/or having been arrested, whereas the current study examined continuous conduct symptoms. Such methodological differences may account for the differing results between the current findings and that of Hay et al. (2010).

Moreover, one counterintuitive study finding was that exposure to maternal depression during preschool predicted *fewer* youth conduct symptoms during early adolescence. This result was surprising because the literature demonstrates maternal depression predicts more child conduct problems (Goodman et al., 2011). One explanation for this counterintuitive finding is that 75% of all youth did not report any conduct symptoms, reducing variability (65% of youth with ADHD and 85% comparison did not self-report any conduct symptoms). Because the current study examined emergence of conduct symptoms during early adolescence, it is also possible results would differ if conduct symptoms were assessed at a later time point (i.e., after age 14), when conduct problems are more prevalent.

Our study also uniquely demonstrated the moderating effect of child ADHD status on maternal depression and youth depressive outcomes. Results indicated that, whereas total recurrence of maternal depression predicted youth depressive symptoms for all youth, this

relation was significantly stronger among children with ADHD. Hammen's (1991) intergenerational interpersonal stress model of depression largely focuses on interpersonal (e.g. family, parenting, marital) stress and youth social competence as the mechanisms by which depression is transmitted from mother to child. Thus, the relation between total recurrence of maternal depression and youth depressive symptoms may be stronger in children with ADHD due to the documented high levels of conflict and low levels of competence present in families and social relationships of children with ADHD, respectively (Johnston & Mash, 2001; Mikami, 2010). While the current study was not able to test Hammen's (1991) intergenerational model of depression, causal mechanisms, such as interpersonal parent-child relationship stress and youth social difficulties, can be examined in future studies using mediation analyses.

Relatedly, preschool exposure to maternal depression predicted youth depressive symptoms during early adolescence only among children with ADHD. This finding, which controlled for later exposure periods to maternal depression, suggests pathways to depression may begin pre-pubertally for children with ADHD. Further, preschool exposure to maternal depression did not predict youth depressive outcomes for comparison children. Such finding fits with the literature suggesting late maternal depression, rather than early exposure, predicts youth depressive outcomes (Hay et al. 2008; Halligan et al., 2007). Alternatively, another possible explanation is that a small number of comparison children (n=9) were exposed to maternal depression during preschool, so it is likely our study was underpowered to detect such effects in comparison children.

Further, child ADHD status did not moderate the relation between recurrence and timing of maternal depression and youth conduct symptoms. The literature suggests that maternal depression is a non-specific risk factor for a host of negative child outcomes, including both internalizing and externalizing problems (Goodman et al., 2011). Thus, recurrence and timing of

maternal depression may pose the same level of risk for conduct problems during early adolescence in children with or without ADHD. Alternatively, while maternal depression may pose the same risk for children regardless of ADHD diagnosis, the mechanisms by which maternal depression predicts conduct problems may be different in children with and without ADHD. While the current study did not include an examination of mechanisms, potential mediators, such as maladaptive parenting, adverse family environments and deviant peer relationships, should be examined in future studies that examine why maternal depression predicts adverse outcomes in children with and without ADHD.

The current study had several strengths. To our knowledge, this is the first study to examine recurrence and timing of maternal depression in predicting outcomes during early adolescence among children with and without ADHD. Methodological strengths include examining exposure to maternal depression during theoretically relevant developmental periods. We assessed maternal depression yearly using structured diagnostic interviews, and used multiple informants of youth depressive and conduct outcomes. Statistically, we controlled for other periods of exposure to maternal depression when examining the effects of a specific period, allowing us to make conclusions about the independent effects of exposure during each developmental period. We also controlled for factors related to depressive and conduct outcomes based on the literature (i.e. child race, maternal education, family income, child sex, child intelligence).

Study limitations include lack of information about the severity, chronicity and duration of maternal depression. Evidence suggests severity and chronicity of maternal depression uniquely predicted youth internalizing and externalizing outcomes (Hammen & Brennan, 2003). Additionally, we did not have information about maternal depression that occurred in the child's lifetime before age 4. Several studies found that antenatal and postpartum depression predicts

offspring outcomes into adolescence (Pearson et al., 2003; Pawlby et al., 2009). Our lack of data during for these periods does not allow us to examine effects of antenatal and postnatal depression on youth outcomes during early adolescence. Additionally, our sample was comprised mostly of males, which prevented us from examining gender as a potential moderator. Moreover, we were not able to examine psychopathology in the co-parent. Some evidence suggests maternal and paternal psychopathology have interactive effects on youth outcomes (Brennan Hammen, Katz, & Le Brocque, 2002). Our study also had a low rate of conduct symptoms. It is possible if we followed our sample into late adolescence, higher rates of conduct symptoms would emerge. Lastly, we did not examine mechanisms by which exposure to maternal depression during specific time periods impacts youth depressive and conduct outcomes.

Future studies should follow offspring into later adolescence and young adulthood, which are developmental periods marked by their own unique challenges. Moreover, incorporating neurobiological markers, such as genetic variants (Nemoda et a., 2015), would further contribute to our understanding of the development of depression and conduct disorder in children with and without ADHD. Additionally, incorporating other indices of parental psychopathology, such as parental ADHD, substance abuse, and antisocial personality disorder, in both parents is also necessary (Chronis et al., 2003). Lastly, future studies should aim to examine mechanisms that explain the relations between recurrence and timing of maternal depression and youth outcomes, such as parent-child relationship quality, parenting behaviors, youth social functioning, and family environment.

Current study results regarding depressive outcomes inform the optimal timing of targeted interventions for children with ADHD and their mothers. Specifically, study results suggest that interventions for children with ADHD would benefit from an additional treatment

component targeting maternal depression (Chronis-Tuscano et al., 2013), and such interventions would be most beneficial if implemented beginning early in development, such as the preschool period (Chronis-Tuscano et al., 2014). Given the recurrent nature of adult depression, implementing such interventions over a longer term may be necessary to reinforce acquired skills. Lastly, early and targeted interventions such as these have the potential to not only improve individual lives, but also enhance functioning at the population and societal level. Given the potential of such interventions to curtail suicidality and criminality through blunting the development of comorbid depression and conduct disorder among children with ADHD, the development, evaluation and implementation of such interventions have utmost public health significance.

Tables

Table 1
Participant Demographic Characteristics at Baseline[*]

	% (N) or M(SD)	
	ADHD (n=125)	COMPARISON (n=122)
Child Age		
4 years old	16% (20)	23% (28)
5 years old	44.8% (56)	36.9% (45)
6 years old	39.2% (49)	39.3% (48)
Child Sex		
Male	85.6% (107)	80.3% (98)
Female	14.4% (18)	18.9% (23)
Child Race		
Caucasian	63.2% (79)	65.6 (80)
African American	30.4% (38)	28.7%(35)
Other	6.4% (8)	4.9% (6)
Maternal Education		
High School	33.6% (42)	27% (33)
College	54.4% (68)	56.5% (69)
Graduate School	10.4% (13)	13.9 (17)
Site		
Chicago	45.6% (57)	41% (50)
Pittsburgh	54.4% (68)	58.1% (71)
Maternal Martial Status		
Ever Legally Married[a]	66.4% (83)	77% (94)
Divorced / Separated[b]	24.0% (30)	17.2% (21)
Median Family Income	$22,500-$30,000	$30,000- $40,000
Child Intelligence	*M*=92.0 *SD*=12.7	*M*=104.6 *SD*=13.7
Maternal Depression		
Lifetime maternal depression	38.4% (48)	23.8% (29)
Preschool exposure (child age 4-6)	14.4% (18)	7.4% (9)
Childhood exposure (child age 7-10)	28% (35)	17.2%(21)
Early adolescence exposure (child age 11-14)	32.8%(41)	18.0% (22)
Total Recurrence	*M*=1.22, *SD*=1.79	*M*=0.61, *SD*=1.36
Youth Outcomes		
Youth Depressive Symptoms – Child	*M*=1.98, *SD*=2.4	*M*=.91, *SD*=1.58
Youth Depressive Symptoms – Parent	*M*=1.25, *SD*=2.11	*M*=.39, *SD*=1.41
Youth Conduct Symptoms –Child	*M*=.47, *SD*=.88	*M*=.14, *SD* =.54
Youth Conduct Symptoms – Parent	*M*=.61, *SD*=1.3	*M*=.10, *SD*=.48
Youth Conduct Symptoms – Teacher	*M*=.55, *SD*=1.28	*M*=.13, *SD* =.57

[*] Percentages and numbers are based on participants with available data
[a] To the biological father at some point
b Since the birth of the child

Table 2
Correlations Among All Variables

	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*	*14*	*15*	*16*	*17*
1. Youth Depressive Symptoms - Parent	1																
2. Youth Depressive Symptoms - Youth	0.471*	1															
3. Youth Conduct Symptoms - Youth	.322	.367*	1														
4. Youth Conduct Symptoms - Teacher	.286*	.324*	0.467*	1													
5. Youth Conduct Symptoms - Parent	.550*	.233*	0.329*	0.225*	1												
6. Child Race - African	0.191*	0.311*	0.194*	0.302*	0.166*	1											
7. Child Race - White	-0.214*	-0.327*	-0.272*	-0.284*	-0.153*	-0.878*	1										
8. Mom Education - School	0.049	0.091	0.035	0.138+	0.052	0.059	-0.093	1									
9. Mom Education - College	0.033	0.002	0.015	-0.047	0.008	0.051	-0.008	-0.765*	1								
10. Family Income	-0.168*	-0.215*	-0.171*	-0.340*	-0.161*	-0.409*	0.403*	-0.390*	0.124	1							
11. Child Intelligence	-0.071	-0.256*	-0.108	-0.225*	-0.109	-0.284*	0.283*	-0.176*	0.054	0.460*	1						
12. Child Sex	0.141*	0.114+	0.014	-0.013	-0.024	-0.052	0.034	0.078	-0.027	0.063	0.036	1					
13. Child ADHD Status	0.234*	0.244*	0.221*	0.209*	0.258*	0.016	-0.030	0.069	-0.027	-0.194*	-0.432*	-0.062	1				
14. Preschool Exposure	0.104	0.186*	0.027	-0.014	0.100	0.170*	-0.175*	-0.022	0.023	-0.162*	-0.237*	-0.017	0.113+	1			
15. Childhood Exposure	0.258*	0.154*	0.289*	0.248*	0.182*	0.113+	-0.145*	0.016	0.038	-0.311*	-0.181*	0.068	0.127*	0.275*	1		
16. Early Adolescence Exposure	0.449*	0.300*	0.277*	0.314*	0.356*	0.176*	-0.136*	0.021	0.061	-0.328*	-0.214*	0.008	0.171*	0.221*	0.420*	1	
17. Total Recurrence	.435*	.324*	.269*	.211*	.280*	.201*	.186*	.028	.047	-.354*	-.277*	.081	.189*	.547*	.727*	.739*	1

* $p<.05$
+ $p<.10$

Table 3
Summary of Results

	Youth Depressive Symptoms			Youth Conduct Symptoms		
	B(SE)	b (SE)	R^2	B(SE)	b (SE)	R^2
Hypothesis 1						
Total Recurrence	**.469(.072)****	**.381(.072)**	**.479****	**.272(.081)****	**.084(.027)****	**.379****
Hypothesis 2						
Preschool Exposure	.029(.076)	.123(.325)	**.500****	**-.178(.078)***	**-.279(.124)***	**.486****
Childhood Exposure	.020(.082)	.063(.264)	**-**	**.218(.084)***	**.256(.104)***	-
Early Adolescence Exposure	**.487(.076)****	**1.46(.276)****	**-**	**.310(.084)****	**.346(.096)****	-
Hypothesis 3						
Total Recurrence X ADHD	**.290 (.127)***	**.267(.117)***	**.504****	.186(.133)	.066(.048)	**.387****
Hypothesis 4						
Preschool X ADHD	**.255(.120)***	**1.31(.619)***	**.520****	.016(.901)	*.029(.237)*	**.487****
Childhood X ADHD	.128 (.117)	.488(.442)	**.507****	.154(.119)	*.217(.170)*	**.493****
Early Adolescence X ADHD	.128(.122)	.457(.435)	**.505****	.052(.129)	*.067(.168)*	**.488****

*Note. N=247; p<.001** ; p<.05*

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