Published in final edited form as:

Dev Psychopathol. 2010 November ; 22(4): 803–818. doi:10.1017/S0954579410000477.

Influence of Parental Depressive Symptoms on Adopted Toddler Behaviors: An Emerging Developmental Cascade of Genetic and Environmental Effects

Caroline K. Pemberton,
The Pennsylvania State University

Jenae M. Neiderhiser,
The Pennsylvania State University

Leslie D. Leve,
Oregon Social Learning Center

Misaki N. Natsuaki,
University of California, Riverside

Daniel S. Shaw,
University of Pittsburgh

David Reiss, and
Yale Child Study Center

Xiaojia Ge
University of Minnesota

Abstract

This study examined the developmental cascade of both genetic and environmental influences on toddlers' behavior problems through the longitudinal and multi-generational assessment of psychosocial risk. We used data from the Early Growth and Development Study, a prospective adoption study, to test the intergenerational transmission of risk through the assessment of adoptive mother, adoptive father, and biological parent depressive symptoms on toddler behavior problems. Given that depression is often chronic, we control for across-time continuity and find that in addition to associations between adoptive mother depressive symptoms and toddler externalizing problems, adoptive father depressive symptoms when the child is 9-months of age were associated with toddler problems and associated with maternal depressive symptoms. Findings also indicated that a genetic effect may indirectly influence toddler problems through prenatal pregnancy risk. These findings help to describe how multiple generations are linked through genetic (biological parent), timing (developmental age of the child), and contextual (marital partner) pathways.

Keywords

child psychopathology; genetics; adoption designs; behavior problems; parental depression; toddler

It is clear that children of depressed parents are at an increased risk for a range of problem behaviors, including externalizing and internalizing symptoms and disorders (Beardslee, Bemporad, Keller, & Klerman, 1983; Bierderman, Faraone, Hirschfeld-Becker, Friedman, Robin, & Rosenbaum; Cummings & Davies, 1994; Goodman & Gotlib, 1999; Orvaschel, Walsh-Allis, & Ye, 1988; Radke-Yarrow, 1998; Weissman et al., 1987). The intergenerational transmission of risk from parental depression to child problem behavior could occur through multiple pathways. Risk could be conferred through an interplay of genes, parenting, and environmental context that shifts and changes across development. It is unlikely that shared genes or environment either fully account for the transmission of risk, which is more likely due to a cascade of multiple effects that influence one another over time (Masten et al., 2005). This paper explores the unfolding of risk over time from parental depressive symptoms to child problem behaviors. We explore the cascade of risk from when children are 9 to 27 months of age, exploring how the timing and source of influence contributes to child problem behaviors at time during which there is rapid individual change and development (see Brownell & Kopp, 2007).

Specific differences in parenting behaviors have been found between depressed and nondepressed parents, with depressed parents tending to be less engaged, less responsive, and less communicative with children than parents with fewer symptoms (e.g., Bettes, 1988; Feng, Shaw, Skuban, & Lane, 2007; Rowe, Pan, & Ayoub, 2005; Shaw et al., 2006). It is challenging, however, to infer that the impact of these parenting differences on child outcomes is due to primarily environmental processes in studies of only biologically-related families because children share both genes and environments with their biological parents. In studies of biological families, mental health problems in children of depressed parents could be due to shared genes or child-driven differences in parenting, not necessarily to parenting differences related to the parents' depression. Moreover, parenting changes across development; parenting behaviors needed for a 9-month-old infant differ from those most appropriate for a 27-month-old toddler because children of different ages have different needs. For instance, as children develop from 9 to 27 months they become more autonomous and acquire language (Gleason, 2005), as well as begin to regulate emotions in accordance with parental expectations (Kopp, 1989). However, these are also ages marked by changes and peaks in high emotional negativity (Bridges, 1932; Campbell, Spieker, Burchinal, & Poe, 2006; Tremblay & Nagin, 2005). As children develop, so too must parenting behaviors, which could be differently influenced by elevated depressive symptoms. In addition, the developing child may be influenced by parents' depressive symptoms at some stages in development more than others, and some children may be more sensitive than others. The developing neurological and attentional systems of children may also differ in their sensitivity to the continuum of parental depressive symptoms. For example, a recent set of papers advances a theory of differential susceptibility such that some children may be differentially susceptible to both positive and negative environmental influences (Belsky &

Pluess, 2009). Some children may be more malleable to environmental influences than other children, and thus having a parent with a low or high number of depressive symptoms may influence some children more than others. Using an adoption design, this study is a first step toward identifying genetic and environmental effects of parental depressive symptoms and how their timing influences child functioning in early childhood.

Depression is a common, debilitating, and recurring disorder (Üstün, Ayuso-Mateos, Chatterji, Mathers, & Murray, 2004; Weissman et al., 1999). Throughout this paper, the term parental depression will be used to describe those who have met clinical criteria for both unipolar depression and those who obtain elevated scores on depressive symptoms inventories. Associations between parental functioning and child outcomes have been documented in both cases (e.g., Cummings, Keller, & Davies, 2005; Anderson & Hammen, 1993). Depression could arguably be especially debilitating in parents of young children, given that caregivers of toddlers are likely to face challenges each day that require interest and energy, and because the parenting of toddlers can benefit from positive emotions and feelings of efficacy (e.g., Dix, 1991; Teti & Gelfand, 1991). Moreover, depression is heritable (Lesch, 2004), with heritability estimated to account for 36% of the variance (Sullivan, Neale, & Kendler, 2000), suggesting that some child behavior and emotional problems related to parental depression may be due to shared genes. Genetically-informed designs can take genetic contributions into account, thus providing an estimate of environmental influences independent of genetic influences. This study aims to advance our knowledge of both the genetic and environmental contributions by examining the timing of adoptive parental depressive symptoms and whether there is a direct genetic influence of parental depressive symptoms on toddler externalizing symptoms. Finding a direct genetic influence would have implications for future research and the ways in which children shape their own environments and outcomes over time, even at such a young age. Understanding the timing of environmental effects of parental depressive symptoms in this context, while also accounting for the chronicity and stability of symptoms over time, is a crucial area of study because this knowledge can inform and improve prevention and intervention efforts. It is possible that the impact of parental depression on child functioning differs depending on the age of the child and/or the developmental stage, and that these times of differential influence could represent sensitive periods of parental influence. However, we do not yet know *when* parental depressive symptoms are most crucial to child development.

Timing of Depressive Symptoms

Goodman and Gotlib (1999) assert that the earlier a child is exposed to parental depression, the more likely that depression will have an impact on the child. They support this hypothesis with evidence detailing the crucial development of inhibitory systems and the HPA system during infancy (Dawson, 1994; Stansbury & Gunnar, 1994; Porges et al., 1994) and with the logic that earlier detriments will have a greater effect on a child's developmental trajectory than later detriments (Hay, 1997). Empirical research is mixed as to whether concurrent or earlier depression influences children the most. For example, previous work has found differential effects of early (infancy) and remitting parental depression versus current-only (preschool) depression (Alpern & Lyons-Ruth, 2003), while one meta-analysis of parental depression across childhood found that concurrent depression,

as opposed to depression measured at earlier ages, is associated with the most negative child outcomes (Lovejoy et al., 2000). Given that depression is often chronic (Weissman et al., 1999), longitudinal studies that account for both prior and current depressive symptoms are necessary for examining the timing of depression. One method of accounting for the chronicity of depression uses latent growth curve modeling to demonstrate that that chronic parental depressive symptoms are more problematic than decreasing or mild depression (Ashman, Dawson, & Panagiotides, 2008; Gross, Shaw, Burwell, & Nagin, 2009); however, this method does not measure the relative importance of depressive symptoms on child behaviors at each observation. This study will focus on children from infancy to early toddlerhood, a period of rapid change (see Brownell & Kopp, 2007) and high dependence on parental responsiveness. Identifying ages of particular vulnerability to parental depressive symptoms could help in creating targeted groups for prevention and early intervention.

Exploring the timing of influence on child behaviors will help to identify the potential cascading effects of parental depressive symptoms over time. Previous research has demonstrated how child problems and competence modeled over time, accounting for across-time continuity, can elucidate the trajectories of behaviors across domains and potential causal factors (Masten et al., 2005). Moreover, the exploration of how multiple domains influence other domains over time has provided a complex theory of the development of substance-use onset (Dodge, Malone, Lansford, Miller, Pettit, & Bates, 2009). These studies provide evidence for the benefit of longitudinal research across multiple domains. In this study, we look at risk from one domain, genetic risk from elevated depressive symptoms in the nonrearing birth parent, while also examining the unfolding effects over time of another domain, the timing of rearing parents' depressive symptoms on child externalizing problems. We explore the emerging cascade of risk across domains and over time.

Mothers and Fathers

Based on the higher prevalence rates of depression in women than men, it is understandable that the majority of studies conducted with parents with depressive symptoms have examined depression in mothers only. Most studies examining maternal depression find that depressed mothers parent more negatively (Lovejoy et al., 2000), and that child outcomes are worse when mothers are depressed (e.g., Cummings & Davies, 1994). Many studies including paternal depression are correlational in nature, although a meta-analysis of these studies suggests significant associations between paternal depression and many types of childhood problem behavior (Kane & Garber, 2004). Multiple studies have shown that children with two depressed parents have an increased risk of behavior problems when compared to children with only one depressed parent (e.g., Brennan, Hammen, Katz, & Le Brocque, 2002; Goodman, Brogan, Lynch, & Fielding, 1993; Lieb, Isensee, Höfler, Pfister, & Wittchen, 2002; Weissman et al., 1987). While recent research highlights importance of including fathers in research (e.g., Gross, Shaw, Moilanen, Dishion, & Wilson, 2008), little is known about the reasons for increased risk. One reason may be that a child who has two parents with depression may be at a greater risk, for genetic reasons, to become depressed themselves (Goodman & Gotlib, 1999). In addition, a depressed father could alter the

childhood environment directly through his parenting or through an exacerbation of the mother's depression. In support of this idea, one study found that high paternal depressive symptoms exacerbated maternal depressive symptoms in infancy, but only in the presence of high child involvement (Mezulis, Hyde, & Clark, 2004). One marital partner may also buffer the effects of the other partner's depression. For example, in an adolescent sample, a positive father-child relationship counteracted the effects of maternal depressive mood on adolescent poor functioning (Tannenbaum & Forehand, 1994). These findings highlight the need to study both mothers and fathers using methodologies that allow genetic and environmental effects to be disentangled.

Using a cascades model, we examine how the environmental effects of having a parent with elevated depressive symptoms influences children while considering genetic contribution. Moreover, we begin to unpack different aspects of the environment, such as considering the influences of mothers and fathers separately and considering them together. While the current study does not explore the mediating factors involved in how mothers and fathers may separately and together influence children over time, it is a first step looking at their influences in early childhood in a genetically-informed way.

Genetically-Informed Designs

Based on the sizable literature underscoring the heritability of depression (e.g., Sullivan et al., 2000) and a similarly large literature on the importance of rearing environment on children's emotional development (e.g., Davis & Windle, 1997; Eisenberg, Cumberland, & Spinrad, 1998; Rutter, 2000), characterizing different patterns of risk on both maternal and paternal depressive symptoms is essential for advancing our understanding of how risk is conferred. The current study uses an adoption design, which allows us to parse genetic from environmental influences. Children placed for adoption at birth or soon after share only genes (and prenatal environment for birth mothers) with their birth parents. Thus, associations between an adopted child and their birth parents' characteristics can be used to estimate genetic contributions to the child's characteristics. Adoptive parents are genetically unrelated to the adopted child, and thus associations between adoptive parents and their child cannot be the result of shared genetic influences between parent and child. Associations could be the result of evocative genotype-environment correlation (rGE), when children evoke a response from their environment. For example, an infant who is genetically predisposed to be fussy may evoke a type of parenting that is different from an infant who is genetically predisposed to less fussy (Natsuaki et al., in press). Associations could also be a direct result of the environment (E), for example, when a parent's over-reactive parenting style leads the child to become fussy over time.

Several studies using genetically-informed designs have demonstrated both genetic and environmental influences of parental depression on child functioning. For example, in a twin sample, family conflict and genetic factors were found to be associated with depression in youth ages 8 to 17 (Rice, Harold, & Thaper, 2002). Few adoption studies have examined the genetic and environmental influence of parental depression on child outcomes. One exception is a study of adoptees that used retrospective reports and records of a history of treatment for depression and/or mania (Cadoret, O'Gorman, Heywood, & Troughton, 1985).

The authors found a positive trend of birth parent depression on young adult depression, but as one meta-analysis pointed out (Sullivan et al., 2000), this study was underpowered and split the results by gender of the adoptee. When Sullivan and colleagues combined the adoptees into one sample, they found a significant genetic effect between biological parent risk and youth depression. In another study, adoptive mother, but not adoptive father, lifetime depression elevated adolescent risk of psychopathology (Tully, Iacono, & McGue, 2008). The effects of birth parent depression and the timing of the depressive symptoms were not tested in this study.

Genetically-informed designs can also examine how parental depression moderates genetic risk. Using the current study's adoptive sample, one study found that children of birth mothers with major depressive disorder are fussier at 18 months when adoptive mothers were less responsive at 9 months of age (Natsuaki et al., in press). Children of adoptive parents with higher levels of responsiveness did show elevated fussiness at 18 months when birth mothers had major depressive disorder. This study demonstrated how children with a genetic risk from major depression may be more vulnerable to certain types of parenting in infancy. This study also found that children whose adoptive parents had more depressive symptoms were fussy, demonstrating an environmental effect. Another study with the current sample found that structured parenting was beneficial to toddlers at genetic risk for psychopathology, but was associated with behavior problems in toddlers at low genetic risk (Leve et al., 2010). Children's reaction to the same type of parenting was dependent on the child's genetic risk. These studies highlight the importance of examining the genetic contributions to child outcomes because they aid in better understanding the effects of parenting behaviors and the environment.

Current Study

Participants are from a longitudinal, prospective adoption study (Early Growth and Development Study, EGDS; Leve et al., 2007; Leve, Neiderhiser, Scaramella, & Reiss, 2008). This study adds to previous findings from EGDS by examining adoptive parental depressive symptoms across time, when the child is 9, 18, and 27 months of age. We also include multiple assessments of biological parent depression. We focus on predicting toddler externalizing problems at 27 months because parents are more accurate reporters of young child externalizing problems than internalizing (e.g., Edelbrock, Costello, Dulcan, Conover, & Kala, 1986; Loeber, Green, Lahey, & Strouthamer-Loweber, 1989; Welner, Reich, Herjanic, Jung, & Amado, 1987) and because continuity with later problem behavior has been found to be stronger for externalizing than internalizing problem behavior from the toddler to school-age periods (Feng, Shaw, & Silk, 2008; Shaw, Gilliom, Ingoldsby, & Nagin, 2003). The current study tests the intergenerational transmission of risk, in essence the cascading of risk, through the longitudinal assessment of adoptive mother, adoptive father, and biological parent depressive symptoms, controlling for across-time continuity, from both genetic and environmental sources. Our focus is on identifying emerging evidence of a "cascade" of effects (Masten et al., 2005), that is a dynamic and transactional developmental process of how genetic and environmental risk factors influence behavior problem development. Evidence of both genetic and environmental influences across time

would suggest a need to incorporate these multiple sources of influence and change in future models seeking to understand causes of change and targets for prevention and intervention.

We expected that a) higher levels of biological parent depressive symptoms would be associated with higher levels of toddler externalizing problems, and b) both earlier and concurrent adoptive mother and father depressive symptoms would be associated with toddler externalizing problems. Given the relative lack of research on father depressive symptoms, we did not make any a priori predictions about differences between mothers and fathers, but did expect adoptive paternal depressive symptoms to account for independent variance in relation to child externalizing problems.

Method

Study Overview and Participants

Participants are from a longitudinal, multisite study: The Early Growth and Development Study. The sample consisted of 361 linked sets of adopted children, adoptive mothers (AM), adoptive fathers (AF), birth mothers (BM), and birth fathers (BF). The analytical sample consisted of 351 linked sets after removing outliers. In addition, 18 of the 351 families were same-sex couples. We systematically tested differences in depressive symptoms, child externalizing problems, and the relations between depressive symptoms and toddler problems between same-sex and opposite-sex couples, and found no significant differences. Therefore, same-sex families were included in the analyses. Only a subsample of the BFs was recruited ($N=95$), and thus BF data were not included in the primary analyses and were tested separately.

Recruitment took place between 2003 and 2006, starting with the recruitment of adoption agencies ($N = 33$ agencies in 10 states located in the Northwest, Mid-Atlantic, and Southwest regions of the United States). Agencies were selected to reflect the full range of adoption agencies in the United States: public, private, religious, secular, those favoring closed adoptions, and those favoring open adoptions. An agency-appointed liaison assisted with recruitment and identified participants who completed an adoption plan through their agency and met the following eligibility criteria: 1) the adoption placement was domestic, 2) the baby was placed within three months postpartum, 3) the baby was placed with a nonrelative adoptive family, 4) the baby had no known major medical conditions such as extreme prematurity or extensive surgeries, and 5) the birth and adoptive parents were able to read or understand English at the eighth-grade level. The liaison used the contact information provided by the agency to invite the birth mother and adoptive parents to participate. The liaison then recruited the birth father or he was recruited through the birth mother. The study participants were representative of the adoptive and birth parent populations that completed adoption plans at the participating agencies during the same time period. See Leve et al. (2007) and Leve et al. (2008) for more details about the study design and sample description.

Forty-three percent of the children were female. The mean child age at placement was 3 days ($SD = 13$ days). The birth parents typically had high school or trade school education levels and household incomes under \$25,000, and adoptive parents were typically college-

educated, middle-class families. Adoptive parents had been married an average of 11.8 years ($SD = 5.1$ years; Range: 1.2 to 27.1 years). Please see Table 1 for additional demographic information about this sample. There were no significant differences between this analytic sample and the full EGDS sample.

Study Design

Birth parent and adoptive family in-person assessments were conducted by interviewers who completed a minimum of 40 hours of training, including a 2-day group session, pilot interviews, and videotaped feedback prior to administering interviews with study participants. All birth parent assessments were conducted in a location convenient to the participant, most often at home. The first birth parent interview occurred 3-6 months (average of 4 months) postpartum and the second was at 18 months. Adoptive family assessments occurred at 9, 18, and 27 months postpartum. All in-person assessments lasted two-three hours and included a collection of mailed self-report booklets, computer-assisted personal interview questions, and interviewer impressions. The first birth parent assessment also included a pregnancy history calendar about the birth mother's drug use, mental health and stress during pregnancy, and each adoptive family assessment included a series of videotaped interaction and standardized tasks.

Measures

Parent depressive symptoms—Depressive symptoms were measured for birth parents at the first two measurement occasions and for adoptive parent depressive symptoms at all three measurement occasions using the Beck Depression Inventory (BDI; Beck & Steer, 1993). Depressive symptoms were calculated as the sum of 20 items from the 21-item BDI (BM $\alpha = .92, .90$; BF $\alpha = .89, .87$; AM $\alpha = .71, .79, .84$; AF $\alpha = .75, .81, .84$); the suicidal ideation item was not administered to minimize situations where clinical follow-up would be needed (Leve et al., 2010).

At approximately 4 months and 18 months postpartum, BMs were also asked over the phone about prenatal depression using selected items from the BDI in the form of a Pregnancy History Calendar. The Pregnancy History Calendar is based on the Life History Calendar method, a strategy that has been shown in prior research to improve retrospective reporting by anchoring the retrospective reporting during specific periods with other significant events occurring during those periods (Caspi et al., 1996). BMs reported on symptoms of depression during the 3 trimesters of their pregnancy. Preliminary analyses also examined full BDI scores and found comparable results.

Toddler behavior problems—At 27 months, AMs and AFs completed the Child Behavior Checklist (Achenbach, 1992), which consists of 99 behaviors rated on a three-point scale with values of 0 (not true), 1 (sometimes true), and 2 (very true). The externalizing problems T score, which sums 24 out of the 99 items, was used in the present analyses (AM $\alpha = .87$; AF $\alpha = .90$), thereby indicating risk for externalizing psychopathology. To best account for severity of child behavioral problems and both mother and father contributions, the highest externalizing CBCL score was used to represent child

problems, regardless of whether taken from mother or father. AM and AF reports of child externalizing scores were correlated at .40 ($p < .001$).

Additional covariates—Several additional covariates were included to control for variables that could confound or result in misinterpretation of the findings.

Openness in adoption: To control for similarities between birth and adoptive families resulting from contact and knowledge between parties, we controlled for the level of openness in the adoption. The perceived openness of APs and BPs, rated on a 7-point scale, were combined to create an aggregated openness measure. There was a high rate of convergence among reporters and the openness measure represents an aggregation across reporters (Ge et al., 2008).

Child sex: Child sex was coded as 1 (*female*) or 2 (*male*).

Parent age: BM, AM, and AF age were entered as control variables.

Prenatal complications: The McNeil-Sjostrom Scale for Obstetric Complications (McNeil & Sjostrom, 1994) was used to create an index of pre- and neo-natal complications. Items included three separate indices: 1) Toxin exposure (exposure to radiation, x-ray, lead, and chemicals), 2) Drug and alcohol use (cigarettes, second-hand cigarette smoke, alcohol, marijuana, cocaine, amphetamines, heroin, prescription pain killers, inhalants, sedatives, and tranquilizers), and 3) Pregnancy risk (circulatory problems, infections, maternal age, prenatal care, weight gain and loss, nausea, bleeding, and fetal movement). We included this variable in analyses in an effort to disentangle genetic influences from those of the prenatal environment. The scale generated a score for each birth mother based on retrospective self-report of pregnancy and neonatal events. Events meeting a minimum threshold of being at least potentially harmful or relevant to infant outcome were summed to form a total score. The McNeil-Sjostrom Scale has been shown to have predictive validity (Nicodemus et al., 2008) and to be more sensitive than other common measures of obstetric complications (McNeil, Cantor-Graae, & Sjostrom, 1994).

Elliot Youth Questionnaire Revised: At approximately 4 months post-birth, BPs were administered the Elliot Youth Questionnaire to measure engagement in various delinquent behaviors (Elliot & Huizinga, 1983). BPs were asked how many times he/she has done any of the law-violating behaviors during a specific time frame (e.g., purposely damaged or destroyed property, purposely set a fire, carried a hidden weapon, stolen something). Index offences (IO) and minor delinquency (MD) subscales were used in this study as an index of BP antisocial behaviors (BM IO $\alpha = .75$; BM MD $\alpha = .84$; BF IO $\alpha = .72$; BF MD $\alpha = .87$).

Personality Diagnostic Questionnaire: At 9 and 18 months, AMs and AFs completed the Personality Diagnostic Questionnaire to assess risk for Antisocial Personality Disorder as defined in the DSM-IV (PDQ; Hyler, 1994). The original True/False scale was adapted into a 4-point scale in order to capture low frequencies of antisocial behaviors. At 9 months a sum of 8 questions about delinquent behaviors was used (AM $\alpha = .75$, AF $\alpha = .82$), and at

18 months 4 additional items adapted for this study were added to the original 8 (AM $\alpha = .75$, AF $\alpha = .89$).

Infant Characteristics Questionnaire: At 9 months, AMs and AFs completed an abridged version of the 24 month Infant Characteristics Questionnaire (ICQ; Bates, Freeland, & Lounsbury, 1979), which consists of 24 items rated on a seven-point scale with values of 7 indicating a very difficult temperament. The fussiness scale, which sums 6 out of the 24 items, was used in the present analyses (AM $\alpha = .81$; AF $\alpha = .82$). Parallel to our measurement of child externalizing problems with the CBCL, the highest fussiness scale was used to represent child fussiness, regardless of whether taken from mother or father. AM and AF reports of child fussiness scores were correlated at .67 ($p < .001$).

Results

Overview of Analyses

We hypothesized higher birth parent depressive symptoms would be associated with higher levels of toddler externalizing problems and that both earlier and concurrent adoptive mother and father depressive symptoms would be associated with toddler externalizing problems. We were specifically interested in the timing of parental depressive symptoms, expecting that both early and concurrent parental depressive symptoms would matter for child externalizing problems during the toddler period. Due to the lower number of BF participants, we used BMs only in our structural equation models to account for genetic effects. BFs were tested in separate post-hoc regressions.

Before completing a full model including adoptive parents, we first examined whether depressive symptoms in BM should be included as multiple assessments in one latent factor or as independent predictors over time. One possibility was that depressive symptoms during pregnancy would be predictive, suggesting the possibility of a prenatal effect. Based on literature suggesting that chronicity of parental depression was as important, if not more important, than having elevated symptoms at any one time point (e.g., Gross et al., 2009), we decided to combine all assessment points to generate a latent factor of BM depressive symptoms. This decision was corroborated by preliminary structural equation analyses suggesting that prenatal depressive symptoms were relatively stable over time and significantly loaded onto the latent factor. In addition, when tested as individual observed variables, the assessments did not differ in their predictions of toddler externalizing problems. BM depressive symptoms were not limited to a prenatal effect, and we combined all assessments to form a latent factor of BM depressive symptoms.

Similarly, before testing the full model including BMs, AMs, and AFs, we first examined the timing effects in AMs and AFs separately in the presence of a genetic effect. Several steps were taken to understand the timing of adoptive parents' depressive symptoms. First, a model was tested in which all three measurements of depressive symptoms were predictive of child externalizing symptoms at 27 months in addition to the genetic prediction (see Figure 1). Second, each measurement was tested alone to determine if there were unique effects of any one measurement. Third, if there were not unique effects of any one

measurement, a latent factor of parental symptoms was created (see Figure 2). The timing effects from these models were used to guide the construction of the full model.

The full model included prediction of child externalizing problems at 27 months by a latent factor of BM depressive symptoms, as well as the determined timing effects of APs. The full model allowed us to understand how parental depressive symptoms influence child externalizing problems in the presence of multiple predictors that may interact with one another. In order to understand how the predictors may interact, several follow-up analyses were planned.

Planned follow-up analyses—We expected several questions to remain following the testing of the full model. First, we were interested in understanding the potential pathways of genetic influence. For example, could the genetic effect operate through prenatal risk? It is also possible that a genetic effect could be associated with early child temperament. For these reasons, we planned to test whether effects of BM depressive symptoms operated through prenatal risk, and whether they predicted child temperament at 9 months. If we found an effect through child temperament, this could also suggest potential evocative gene-environment correlation through relations between child temperament and parenting. Last, depending on the AP timing effects, we planned to test whether one parent's depressive symptoms could influence the other's, thus better accounting for the larger family context and interrelationships.

Descriptive Data

The means, standard deviations, ranges, minimums, and maximums for variables of interest are presented in Table 2. Log transformations and square root transformations were used to improve skewed distributions on all AM and AF depressive symptoms variables. Each model controls for parental antisocial behaviors. In addition child fussiness at 9 months, openness of the adoption process, BM age, AM age, AF age, child sex, pregnancy risk, prenatal exposure to toxins, and prenatal exposure to drugs were tested as controls.

Structural equation models were executed with covariance matrices of observed variables, using THEIL (Molenaar, 1996), a FORTRAN program for robust covariance matrix estimation. It improves the condition of a covariance matrix while leaving its underlying structure intact. Descriptives, correlations, and data preparation were calculated in SPSS 18.0. Modeling was performed using LISREL 8.80 (Jöreskog & Sörbom, 1996).

Model Fit

Model fit was assessed using the root mean square error of approximation (RMSEA), the comparative fit index (CFI), and the Normal Theory Weighted Least Squares Chi-Square (χ^2). The RMSEA index includes adjustments for model complexity and is less influenced by the number of parameters in the model than other indicators of fit. The CFI compares the covariance matrices predicted by the model to the observed covariance matrix, and compares the null model with the observed covariance matrix. Like RMSEA, CFI is among the measures least affected by sample size (Fan, Thompson, & Wang, 1999). For RMSEA, a value less than .05 is considered a good fit, and below .08 an adequate fit (Kline, 2004). By

convention, the CFI should be equal or greater than .90 to accept the model. Chi-square is influenced by sample size and indicates good fit when $p > .05$. AIC was used to compare models; A lower AIC indicates a better fit. Only standardized betas are reported.

AM and BM Depressive Symptoms and Toddler Externalizing Problems

In this model, both AM and BM depressive symptoms were examined as predictors of toddler externalizing problems. We first tested a model that included all hypothesized paths of interest and covariates. These included paths from AM symptoms at 9, 18, and 27 months and from the latent BM depressive symptoms variable to 27 month toddler externalizing problems. In this model, only one covariate, child 9 month fussiness, significantly predicted toddler externalizing symptoms. In addition, AM antisocial behavior at 9 months ($\beta = .20, p < .05$), and marginally at 18 months ($\beta = .07, p < .10$), predicted AM depressive symptoms. BM antisocial behavior significantly predicted BM depressive symptoms ($\beta = .38, p < .05$). The model was not an adequate fit to the data (AIC = 383.58, RMSEA = .054; CFI = .88; $\chi^2(146) = 295.58, p < .05$). As expected, AM depressive symptoms were moderately stable over time (β 's = .56, .37, .31). In this full model with covariates and all hypothesized paths, only one hypothesized path to child externalizing problems was marginally significant: BM depressive symptoms ($\beta = .11, p < .10$).

In order to understand the AM timing effects to be entered into the full model, insignificant covariates predicting toddler externalizing problems were removed and each assessment of AM depressive symptoms (9, 18, or 27 months) was tested systematically. AM and BM antisocial behaviors were included in this model and significantly predicted parent depressive symptoms. While controlling for child temperament at 9 months, each assessment, when the other two assessments were removed, significantly predicted child externalizing problems at 27 months. Consequently, we decided to combine all assessments into a latent factor. This model with the latent factor, although still not an adequate fit, was an improvement over the previous model (AIC = 288.22, RMSEA = .10; CFI = .77; $\chi^2(51) = 234.22, p < .05$). In this model, the latent factor of AM depressive symptoms significantly predicted toddler externalizing problems ($\beta = .16, p < .10$). BM depressive symptoms were also significantly related ($\beta = .11, p < .10$).

AF and BM Depressive Symptoms and Toddler Externalizing Problems

AF and BM depressive symptoms were examined as predictors of toddler externalizing problems at 27 months of age. As with AMs, we first tested a model that included all hypothesized paths of interest and covariates. This model was not an adequate fit to the data (AIC = 391.35, RMSEA = .055; CFI = .88; $\chi^2(146) = 303.35, p < .05$). As expected, adopted father depressive symptoms were stable over time (β 's = .50, .43, and .37). In this full model, AF depressive symptoms at 9 months of child age was a marginally significant predictor of child externalizing symptoms ($\beta = .11, p < .10$) and BM depressive symptoms was significantly predictive ($\beta = .11, p < .05$). In this model, two covariates were significantly related to toddler externalizing symptoms, 9 month fussiness and pregnancy risk (marginally). In addition, AF antisocial behavior marginally at 9 months ($\beta = .10, p < .10$), and significantly at 18 months ($\beta = .09, p < .05$), predicted AF depressive symptoms. BM antisocial behavior significantly predicted BM depressive symptoms ($\beta = .39, p < .05$).

Similar to our systematic tests of timing effects of AM, we tested each assessment of AF depressive symptoms individually. As indicated by the model with all paths, only 9 month depressive symptoms was significantly predictive of toddler externalizing problems ($\beta = .12$, $p < .05$; AIC = 186.51, RMSEA = .055; CFI = .94; $\chi^2(62) = 128.51$, $p < .05$).

AM, AF, and BM Depressive Symptoms and Toddler Externalizing Problems

We next examined a model that tested BM, AM, and AF predictors together. We used our timing effects indicated by the prior models: BM and AM were tested as latent factors, and AF depressive symptoms at 9 months was included. We also included child fussiness at 9 months and pregnancy risk (see Figure 3). As shown in the figure, we also added a bidirectional path between BM depressive symptoms to pregnancy risk, as we expected that these two constructs would be related. As in previous models, we controlled for the effects of AP and BM antisocial behavior on depressive symptoms, and these significant associations followed the same pattern as reported in the above models.

The model fit was acceptable by the RMSEA and CFI indices (AIC = 299.02, RMSEA = 0.043; CFI = 0.95; $\chi^2(126) = 209.02$, $p > .05$). The pattern of findings follows those of above: AM depressive symptoms ($\beta = .14$, $p < .05$) and 9 month AF depressive symptoms ($\beta = .11$, $p < .05$) were significantly associated with toddler externalizing problems at 27 months of age. BM depressive symptoms were marginally associated with toddler externalizing problems at 27 months of age ($\beta = .09$, $p < .10$), and were significantly correlated pregnancy risk ($r = .22$, $p < .05$). Pregnancy risk was not significantly associated with toddler externalizing and child fussiness at 9 months was significantly predictive ($\beta = .29$, $p < .05$). Child fussiness at 9 months was allowed to correlate with AM depressive symptoms and 9 month AF depressive symptoms ($r_s = .13$ and $.14$, respectively).

Planned Follow-Up Analyses: Exploring Genetic, Evocative, and Timing Effects

We had several planned analyses following the analyses above. First, we wondered whether the genetic effect of BM depressive symptoms was operating through pregnancy risk. Second, we tested whether there was an association between BM depressive symptoms and child fussiness that would reveal a genetic effect through child temperament or an evocative gene-environment correlation through relations between child temperament and parenting. Last, we examined possible associations between APs, specifically whether early father depressive symptoms at 9 months were associated with AM depressive symptoms.

Genetic effect through pregnancy risk—We removed the direct path from BM depressive symptoms to child externalizing symptoms at 27 months and added a direct path from BM depressive symptoms to pregnancy risk to examine the indirect genetic effect through BM pregnancy risk. BM depressive symptoms significantly predicted pregnancy risk ($\beta = .25$, $p < .05$) and pregnancy risk significantly predicted toddler externalizing problems ($\beta = .10$, $p < .05$). This model also had a lower AIC than the previous model and had an adequate fit as judged by RMSEA and CFI indices (AIC = 296.33, RMSEA = 0.043; CFI = 0.95; $\chi^2(127) = 208.33$, $p < .05$), indicating a better fit. This model suggests that there is an indirect effect of BM depressive symptoms on child externalizing problems at 27 months through pregnancy risk.

Evocative effect of child fussiness at 9 months on AP depressive symptoms—

The significant bi-directional association between fussiness and AP depressive symptoms suggests two possible hypotheses: 1) AP depressive symptoms may indirectly influence child externalizing symptoms at 27 months through 9 months child fussiness, and 2) child fussiness may indirectly influence child externalizing symptoms at 27 months through AP depressive symptoms. The second hypothesis would suggest a potential evocative effect of a biologically-driven fussiness from the child on parents' depressive symptoms, which in turn influence later child externalizing factors. However, tests of unidirectional paths of both hypotheses yielded comparable results, suggesting that we are not able to determine the direction of effects in this analysis.

Timing Effects of APs—In the analyses mentioned above, we found that exposure to AF depressive symptoms when the child was 9 months old was predictive of later child externalizing problems at 27 months of age, whereas AM depressive symptoms as a latent factor composed of all assessments predicted child externalizing problems. In follow-up analyses several steps were taken to see if we could partially explain why the timing of exposure to depressive symptoms differed by the parental status (mother or father) of adoptive parents. We tested the above model with the addition of one path: AF depressive symptoms at 9 months to AM depressive symptoms. The model fit was acceptable as judged by RMSEA and CFI indicators (RMSEA = 0.042; CFI = .96; $\chi^2(126) = 203.25, p = ns$). The AIC of this model (AIC = 293.25) is lower than previous models indicating a better fit. The additional path was significant ($\beta = .14, p < .05$), and none of the other paths meaningfully changed. See Table 3 for a summary of the results and Figure 4 for a depiction of this final model.

BF Linear Regressions

The hypotheses were tested in a parallel manner using BF depressive symptoms as a predictor of child externalizing problems. Due to the reduced sample size of BFs, these analyses were only computed with 22% of the full data, thus requiring a much higher magnitude of correlation to achieve statistical significance. BF depressive symptoms were not predictive of child externalizing problems regardless of whether the 4 month and 18 month BF data were examined independently, or whether a BF depressive composite was examined.

Discussion

This study helps to advance our understanding of how and when parental depressive symptoms contribute to problematic child outcomes, partitioning biological and environmental risk. First, we sought to understand the genetic and environmental effects of having a parent with elevated levels of depressive symptoms. We also used prospective longitudinal data to understand how the timing of parental depressive symptoms could influence the development of early child externalizing problems. We focused on the effects from infancy (9 months) to toddlerhood (27 months). This is the first genetically-informed design that examines genetic, prenatal, environmental, and timing effects of parental depressive symptoms during these crucial years characterized by important transitions in

social, emotional, and cognitive development (Perez & Gauvain, 2007). The results suggest that there is an emerging cascade of risk on toddler externalizing problems, characterized by an indirect genetic effect through prenatal risk and environmental effects that differ depending on timing and parent. This suggests that there are sensitive periods in children's development of externalizing problems that are related to the presence of elevated depressive symptoms in their parents. The findings of this study have implications for identifying toddlers at risk for behavior problems and prioritizing prevention and early intervention for parents with symptoms of depression.

First, in support of previous work (e.g., Sullivan et al., 2000) we found evidence for a genetic effect of depressive symptoms on child disruptive behaviors; however, we found that this effect seemed to operate via pregnancy risk. Birth mothers with more depressive symptoms had more problems during pregnancy, such as attending fewer prenatal care visits, losing/gaining more weight, and maternal illness. In turn, mothers with more pregnancy risk had toddlers with higher levels of externalizing problems at 27 months. This association was significant in the presence of both adoptive mother and adoptive father depressive symptoms, suggesting that there is significant genetic risk from maternal depressive symptoms operating through the prenatal environment that is unique from the postnatal effects of interacting with a caregiver or caregivers struggling with depressive symptoms. To date, most studies reporting associations between parental depressive symptoms and child externalizing and related behaviors have interpreted these associations as a result of disruptions in parenting, family context, and reduced responsiveness or sensitivity on the part of the depressed parent (e.g., Chronis et al., 2007; Cummings, Keller, & Davies, 2005). Although our findings support the important role of parental depressive symptoms as an environmental influence on child functioning, these findings also suggest that at least some of the impact of maternal depression on toddler problems is due to shared genes and the intrauterine environment. Previous findings from the EGDS have supported the roles of both genetics and environment. Nine-month-old infants at genetic risk, as indexed by birth parents' externalizing behavior, showed heightened attention to frustrating events only when adoptive mothers had higher levels of anxious/depressive symptoms (Leve et al., 2010). In this study we find an indirect effect of genetic risk through the prenatal environment in addition to main effects of environmental risk.

It is important that prenatal factors and risk be considered when examining genetic and environmentally mediated effects, particularly in adoption designs in which genetic effects could be at least partially due to the prenatal environment. Multiple studies have found associations between perinatal complications, toxins, and drug and alcohol use during pregnancy and externalizing problems in offspring (e.g., Beck & Shaw, 2005; Brennan, Greken, Mortensen, & Mednick, 2002; Wakschlag, Leventhal, Pine, Pickett, & Carter, 2006). Using a statistical method that controls for environmental and genetic effects using analysis of siblings differentially exposed to alcohol during pregnancy, unique effects of alcohol use were found (D'Onofrio, Van Hulle, Waldman, Rodgers, Rathouz, & Lahey, B.B., 2007). However, a study that controlled for environmental and genetic confounds using the same analytic strategy found much reduced effects of smoking during pregnancy (D'Onofrio, Van Hulle, Waldman, Rodgers, Harden, Rathouz, et al., 2008). Adoption studies with adequate prenatal environment measurement can consider the associations

between biological parent factors, such as depressive symptoms, and the prenatal environment. Consideration of this relationship could uncover possible pathways of genetic influence. In this study, we found that genetically-driven characteristics in birth parents were associated with certain prenatal environments, through which the child was influenced. One large-scale study found significant effects of stress events during pregnancy and smoking during pregnancy on child problems at age two in addition to postnatal risk factors (Robinson et al., 2008), supporting our finding that both pre- and post-natal factors can contribute to child outcomes. Our findings also suggest that the prenatal risk factors were driven by genetic factors in the birth mother.

Second, we found that after accounting for across-time continuity, adoptive mothers' depressive symptoms contribute to toddlers' externalizing problems regardless to the timing of the depressive symptoms during the infancy to toddler period. This may speak to the disparity in the research with biological families, where some research finds earliest depressive symptoms to matter the most (e.g., Alpern & Lyons-Ruth, 2003), whereas others find concurrent parent depression to adversely affect children's behavior in early and middle childhood (e.g., Cummings & Davies, 1994; Goodman & Gotlib, 1999; Radke-Yarrow, 1998), and early intervention research suggesting that improvement in maternal depressive symptoms are associated with decreases in child externalizing and internalizing symptoms during the toddler and preschool periods (Shaw, Dishion, Connell, Wilson, & Gardner, 2009). It may be difficult to parent infants with depressive symptoms, when the increased demands require energy and tolerance. In toddlerhood, mothers with depressive symptoms may struggle to guide their toddler during this age when toddlers need consistent supervision, support, and reinforcement. It is emotionally challenging to raise a toddler (Dix, 1991), and as toddlers gain more language, mobility, and independence, they may be particularly challenging for mothers with more depressive symptoms. Mothers with more depressive symptoms have been found to be less engaged and responsive and to speak less to their children (e.g., Bettes, 1988; Feng et al., 2007; Rowe et al., 2005; Shaw et al., 2006). We can then hypothesize that the mothering behaviors pulled for by an active two-year-old could be especially difficult for a mother who feels fatigued, worthless, or hopeless. Infants and toddlers whose mothers have less energy and availability, and who provide less guidance, emotional support, and consistent consequences may in turn display more externalizing symptoms that are unique of biological attributes shared with their parent.

Third, in contrast to the abovementioned finding regarding the importance of concurrent depressive symptoms in adoptive mothers, we found that adoptive fathers' depressive symptoms at 9 months of child age prospectively contributed to toddler externalizing problems at 27 months of age. To further explore this finding, we tested whether adoptive fathers' depressive symptoms at 9 months were associated with adoptive mothers' symptoms at 27 months, and found a significant association. While this raises the possibility that some of the influence of father depressive symptoms on toddlers is transmitted through the mother, it is notable that father symptoms at 9 months remained a significant predictor of toddler externalizing problems at 27 months, even after accounting for across-time continuity and with mother's depressive symptoms in the model. This is in contrast to previous research indicating that concurrent maternal depressive symptoms accounted for the effects of father depressive symptoms in early infancy (Carro, Grant, Gotlib, & Compas,

1993). While fathers can behave just as sensitively and appropriately in parent-child interactions as mothers (Belsky, Gilstrap, & Rovine, 1984; Lamb, 1997), research on fatherhood and parenting suggests that, on average, fathers become more engaged in child-rearing as the child becomes able to speak and act independently in toddlerhood (Laflamme, Pomerleau, & Malcuit, 2002). One study that examined predictors of mother- and father-infant attachment at one year found that fathers' attitude, but not mothers', toward the infant and parent role predicted the level of attachment (Cox, Owen, Henderson, & Margand, 1992). In early infancy, the mothers' role may be more determined; for example, in Western cultures mothers are more often looked to for feeding, diaper-changing, and soothing (Belsky et al., 1984; Lamb & Oppenheim, 1989).

Perhaps, at 9 months, elevated depressive symptoms are more detrimental to fathers' effects on child outcomes than at later child ages. Fathers who are less sure of their parenting role and have elevated depressive symptoms may be either more withdrawn or more emotionally reactive to their children's unpredictable and labile states in infancy. As the children grow, it may be easier for fathers to interact with their children, even with elevated depressive symptoms. Fathers are also expected to be more playmates in toddlerhood and less managers of basic care (Bretherton, 1985; Laflamme, Pomerleau, & Malcuit, 2002; McBride & Mills, 1993; Tiedje & Darling-Fisher, 1993; Wille, 1995), and their own struggles with depressive symptoms may be less detrimental than mothers' depressive symptoms as mothers are often expected to manage the everyday challenges of raising a toddler.

Some of the literature on parental depression suggests that depressive symptoms may have a greater effect on maternal than paternal parenting (Connell & Goodman, 2002; Field, Hossain, Malphurs, 1999). In contrast, we find direct effects of paternal depressive symptoms on later child externalizing problems. However, this does not delineate the mechanism of transmission. These direct effects could be through paternal parenting or through variables unmeasured in this study. Some research finds that the level of father involvement moderates the effect of maternal depression on child outcomes (Mezulis et al., 2004) or that maternal postpartum depression only contributes to toddler behavior problems if paternal psychopathology is present (Dietz, Jennings, Kelley, & Marshal, 2009). Our finding that paternal depressive symptoms at 9 months relates to maternal depressive symptoms suggests some link between parental symptoms that may develop across child development, marriage, and through mechanisms that were unexplored in this study. For example, depression could alter co-parenting, marital satisfaction (see review by Beach, Fincham, & Katz, 1998), spousal support (Rook, Pietromonaco, & Lewis, 1994), and problem solving skills (Christian, O'Leary, & Vivian, 1994). Future research could benefit from examining mechanisms of risk transmission within a genetically-informed framework and in the family systems context.

Studies that clarify the pathways and timing of risk transmission to young children can inform prevention and early intervention work, as the targeting of parental depressive symptoms may benefit children's own psychopathology (Reyno & McGrath, 2006; Weissman et al., 2006). We found evidence of both genetic and environmental influences of parental depressive symptoms on toddler externalizing problems, and that the timing of symptoms, and perhaps the interaction between parents, could influence early child behavior

problems. Future research should consider additional factors such as parental involvement, attitudes, co-parenting, marital conflict, and marital satisfaction.

Future research should also expand on this emerging cascade by considering both genetic and environmental risk in the prediction of child behavior problems. These findings demonstrate the necessity of considering multiple domains of influence and how their linkages influence one another across time (Masten et al., 2005). These cascades are arguably especially important in infancy and early childhood when children's cognitive, emotional, and physical domains are developing rapidly, and they are highly dependent on caregivers and the environment. This study examines change through toddlerhood, considering genetic influences and the effects of a dynamic and changing caregiver environment.

Limitations

A number of limitations of this study should be considered. First, the EGDS participants are representative of birth and adoptive families from the agencies participating in this study; however, the adoptive families have high educational and economic backgrounds in comparison to national norms. There is a need to include demographic and material-economic factors in studies of parental depression (Oyserman, Mowbray, Meares, & Firminger, 2000). For generalizability to more high-risk environments and more ethnically diverse samples, further studies need to be conducted. The demographic differences between the adoptive and biological parents in terms of age and socioeconomic status, both of which could conceivably contribute to the impact of psychological factors on child outcomes, could also be considered; however, past adoption studies show that the favorability toward adoptive parents has negligible effects on heritability and environmental estimates (McGue et al., 2007).

Second, our genetic findings were not confirmed with depressive symptoms data from birth fathers. Replication of the findings using birth fathers' data would have provided a strong confirmation of genetic effects because the effects from a birth father to a child are not confounded with prenatal environmental factors. Without birth father data we are only including half of the child's genetic makeup. Several methodological issues may have contributed to our non-significant findings in birth fathers' data. First, although EGDS has a high number of birth fathers, the sample is still small for these analyses. Second, the birth fathers have a lower average and smaller range of depressive symptoms than the birth mothers participating in our study (see Table 1), which could limit their predictive power. We know, however, that the observed effects from birth mothers to children are not simply the result of prenatal stress from depression during pregnancy because prenatal depression did not make a prediction to child functioning unique of postnatal birth mother depressive symptoms. Moreover, the aim of this study was not to isolate prenatal risk factors, although evidence influence from pregnancy risk was found. Future work incorporating careful measurement of genes, prenatal environment, and postnatal environment is needed to further disentangle these influences (Knopik, 2009).

Third, we rely on self-report data for parental depressive symptoms, and on adoptive parent report for child externalizing problems. We note that there are potential limitations to the

self-report of symptomatology of depression, and that depressed parents may have biased ratings of their children's problems (e.g., Briggs-Gowan, Carter, & Schwab-Stone, 1996). We attempted to mitigate the biased reports of parents by using the highest behavior problem score of the child from either adoptive mother or father and using this same score in both analyses. We noted that mothers' and fathers' reports of child problems were significantly correlated ($r = .40, p < .001$).

Last, future models could examine reciprocal effects over time, such as one study that found child noncompliance at age 2 to predict concurrent maternal depressive symptoms, which in turn predicted age 4 internalizing and externalizing problems (Gross, Shaw, Moilanen, Dishion, & Wilson, 2008). This study also does not examine a number of potentially important contextual factors such as marital interaction, socio-economic status, social-supports, and siblings. Some of these factors have been shown to be important for the variables in this analysis, for example, there are additive effects for maternal depression and marital conflict on child outcomes (Essex, Klein, Cho, & Kraemer, 2003).

As we begin to refine our understanding of the dynamic and changing relations between parental depressive symptoms, child effects, and child mental health, it will benefit the research to consider genetic and timing effects to best capture the transmission of risk. These effects suggest an emerging cascade, where in the presence of a genetic effect, parental depressive symptoms influence toddlers' behavior at crucial points in development. This cascade can be further explored in the future by include more assessments of child behaviors and exploration of gene-environment correlation and interaction.

Acknowledgments

Data collection for this project was supported by R01 HD042608; NICHD, NIDA, and the Office of the Director; NIH; U.S. PHS (PI Years 1–5: David Reiss; PI Years 6–10: Leslie D. Leve). The writing of this manuscript was supported by the first author's fellowship on T32 MH070327, NIMH, NIH, U.S., PHS (PI: Pamela M. Cole), and was partially supported by R01 DA020585, NIDA, NIMH, and the Office of the Director; NIH; U.S. PHS (PI: Jenae M. Neiderhiser). The content is solely the responsibility of the authors and does not necessarily represent the official views of the Eunice Kennedy Shriver National Institute of Child Health and Human Development or the National Institutes of Health.

We thank the adoptive and birth families who participated in this study, the adoption agency staff members who helped with the recruitment of study participants and Samuel Simmons, Sally Guyer and Kristine Marceau for data management assistance. We would also like to acknowledge the late Xiaojia Ge, close and long time collaborator on this study. Dr. Ge had been invited to contribute to this special issue. His contributions to this study are substantial and we will long feel the loss of our colleague.

References

- Achenbach, TM. Manual for the Child Behavior Checklist/2-3 and 1992 Profile. Burlington, VT: University of Vermont Department of Psychiatry; 1992.
- Alpern L, Lyons-Ruth K. Preschool children at social risk: Chronicity and timing of maternal depressive symptoms and child behavior problems at school and at home. *Development and Psychopathology*. 2003; 5:371–387.
- Anderson CA, Hammen CL. Psychosocial outcomes of children of unipolar depressed, bipolar, medically ill, and normal women: A longitudinal study. *Journal of Consulting and Clinical Psychology*. 1993; 61:448–454. [PubMed: 8326046]

- Ashman SB, Dawson G, Panagiotides H. Trajectories of maternal depression over 7 years: Relations with child psychophysiology and behavior and role of contextual risks. *Development and Psychopathology*. 2008; 20:55–77. [PubMed: 18211728]
- Bates JE, Freeland CAB, Lounsbury ML. Measurement of infant difficulties. *Child Development*. 1979:794–803. [PubMed: 498854]
- Beach SRH, Fincham FD, Katz J. Marital therapy in the treatment of depression: Toward a third generation of therapy and research. *Clinical Psychology Review Special Issue: Behavioral Couples Therapy*. 1998; 18:645–661.
- Beardslee WR, Bemporad J, Keller MB, Klerman GL. Children of parents with major affective disorder: A review. *American Journal of Psychiatry*. 1983; 140:825–832. [PubMed: 6344660]
- Beck, AT.; Steer, RA. *Manual for the Beck Depression Inventory*. San Antonio, TX: Psychological Corporation; 1993.
- Beck JE, Shaw DS. The influence of perinatal complications and environmental adversity on boys' antisocial behavior. *Journal of Child Psychology and Psychiatry*. 2005; 46:35–46. [PubMed: 15660642]
- Belsky J, Gilstrap B, Rovine M. The Pennsylvania Infant and Family Development Project: I. Stability and change in mother-infant and father-infant interaction in a family setting at one, three, and nine months. *Child Development*. 1984; 55:692–705. [PubMed: 6734311]
- Belsky J, Pluess M. Beyond diathesis stress: Differential susceptibility to environmental influences. *Psychological Bulletin*. 2009; 135:885–908. [PubMed: 19883141]
- Bettes BA. Maternal depression and motherese: Temporal and intonational features. *Child Development*. 1988; 59:1089–1096. [PubMed: 3168616]
- Bierderman J, Faraone SV, Hirshfeld-Becker DR, Friedman D, Robin JA, Rosenbaum JF. Patterns of psychopathology and dysfunction in high-risk children of parents with panic disorder and major depression. *American Journal of Psychiatry*. 2001; 158:49–57. [PubMed: 11136633]
- Brennan PA, Grekin ER, Mortensen EL, Mednick SA. Relationship of maternal smoking during pregnancy with criminal arrest and hospitalization for substance abuse in male and female adult offspring. *American Journal of Psychiatry*. 2002; 159:48–54. [PubMed: 11772689]
- Brennan PA, Hammen C, Katz AR, Le Brocque RM. Maternal depression, paternal psychopathology, and adolescent diagnostic outcomes. *J Consult Clinical Psychology*. 70:1075–1085.
- Bretherton I. A sampler of parent-child relations. *PsycCRITIQUES*. 1985; 30:637–638.
- Bridges KMB. Emotional development in early infancy. *Child Development*. 1932; 3:324–341.
- Briggs-Gowan MJ, Carter AS, Schwab-Stone M. Discrepancies among mother, child, and teacher reports: Examining the contributions of maternal depression and anxiety. *Journal of Abnormal Child Psychology*. 1996; 24:749–765. [PubMed: 8970908]
- Brownell, CA.; Kopp, CB., editors. *Socioemotional development in the toddler years: Transitions and transformations*. New York: Guilford; 2007.
- Cadoret RJ, O'Gorman TW, Heywood E, Troughton E. Genetic and environmental factors in major depression. *Journal of Affective Disorders*. 1985; 9:155–164. [PubMed: 2932489]
- Campbell SB, Spieker S, Burchinal M, Poe MD. the NICHD Early Child Care Research Network. Trajectories of aggression from toddlerhood to age 9 predict academic and social functioning through age 12. *Journal of Child Psychology and Psychiatry*. 2006; 47:791–800. [PubMed: 16898993]
- Carro MG, Grant KE, Gotlib IH, Compas BE. Postpartum depression and child development: An investigation of mothers and fathers as sources of risk and resilience. *Development and Psychopathology Special Issue: Milestones in the development of resilience*. 1993; 5:567–579.
- Caspi A, Moffitt TE, Thornton A, Freedman D, et al. The life history calendar: A research and clinical assessment method for collecting retrospective event-history data. *International Journal of Methods in Psychiatric Research*. 1996; 6(2):101–114.
- Christian JL, O'Leary KD, Vivian D. Depressive symptomatology in maritally discordant women and men: The role of individual and relationship variables. *Journal of Family Psychology*. 1994; 8:32–42.
- Chronis AM, Lahey BB, Pelham WE, Williams SH, Baumann BL, Kipp H, Jones HA, Rathouz PJ. Maternal depression and early positive parenting predict future conduct problems in young

- children with Attention-Deficit/Hyperactivity Disorder. *Developmental Psychology*. 2007; 43:70–82. [PubMed: 17201509]
- Connell AM, Goodman SH. The association between psychopathology in fathers versus mothers and children's internalizing and externalizing behavior problems: A meta-analysis. *Psychological Bulletin*. 2002; 128:746–773. [PubMed: 12206193]
- Cox MJ, Owen MT, Henderson VK, Margand NA. Prediction of infant-father and infant-mother attachment. *Developmental Psychology*. 1992; 28:474–483.
- Cummings EM, Davies PT. Maternal depression and child development. *Journal of Child Psychology and Psychiatry*. 1994; 35:73–112. [PubMed: 8163630]
- Cummings EM, Keller PS, Davies PT. Towards a family process model of maternal and paternal depressive symptoms: exploring multiple relations with child and family functioning. *Journal of Child Psychology and Psychiatry*. 2005; 46:479–489. [PubMed: 15845128]
- Davis PT, Windle M. Gender-specific pathways between maternal depressive symptoms, family discord and adolescent adjustments. *Developmental Psychology*. 1997; 3:657–668.
- Dawson, G. Development of emotional expression and emotion regulation in infancy: Contributions of the frontal lobe. In: Dawson, G.; Fischer, KW., editors. *Human behavior and the developing brain*. New York: Guilford Press; 1994.
- Dietz LJ, Jennings KD, Kelley SA, Marshal M. Maternal depression, paternal psychopathology, and toddlers' behavior problems. *Journal of Clinical Child and Adolescent Psychology*. 2009; 38:48–61. [PubMed: 19130357]
- Dix T. The affective organization of parenting: Adaptive and maladaptive processes. *Psych Bulletin*. 1991; 110:3–25.
- D'Onofrio BM, Van Hulle CA, Waldman ID, Rodgers JL, Harden KP, Rathouz PJ, et al. Smoking during pregnancy and offspring externalizing problems: An exploration of genetic and environmental confounds. *Development and Psychopathology*. 2008; 20:139–164. [PubMed: 18211732]
- D'Onofrio BM, Van Hulle CA, Waldman ID, Rodgers JL, Rathouz PJ, Lahey BB. Causal inferences regarding prenatal alcohol exposure and childhood externalizing problems. *Archives of General Psychiatry*. 2007; 64:1296–1304. [PubMed: 17984398]
- Edelbrock C, Costello M, Dulcan M, Conover NC, Kala R. Parent-child agreement on child psychiatric symptoms assessed via structured interview. *Journal of Child Psychiatry*. 1986; 27:181–190.
- Eisenberg N, Cumberland A, Spinrad TL. Parental socialization of emotion. *Psychological Inquiry*. 1998; 9:241–273. [PubMed: 16865170]
- Elliot DS, Huizinga D. Social class and delinquent behavior in a national youth panel. *Criminology: An Interdisciplinary Journal*. 1983; 21:149–177.
- Essex MJ, Klein MH, Cho E, Kraemer HC. Exposure to maternal depression and marital conflict: Gender differences in children's later mental health symptoms. *Journal of the American Academy of Child & Adolescent Psychiatry*. 2003; 42:728–737. [PubMed: 12921481]
- Fan X, Thompson B, Wang L. Effects of sample size, estimation methods, and model specification on structural equation modeling fit indexes. *Structural Equation Modeling*. 1999; 6:56–83.
- Feng X, Shaw DS, Skuban EM, Lane T. Emotional exchange in mother-child dyads: Stability, mutual influence, and associations with maternal depression and child problem behavior. *Journal of Family Psychology*. 2007; 21(4):714–725. [PubMed: 18179343]
- Feng X, Shaw DS, Silk JS. Developmental trajectories of anxiety-related behaviors among boys across early and middle childhood. *Journal of Abnormal Psychology*. 2008; 117:32–47. [PubMed: 18266484]
- Field TM, Hossain Z, Malphurs J. "Depressed" fathers' interactions with their infants. *Infant Mental Health Journal Special Issue: Fathers and infants*. 1999; 20:322–332.
- Ge X, Natsuaki MN, Martin D, Leve LD, Neiderhiser JM, Shaw DS, et al. Bridging the divide: Openness in adoption and post-adoption psychological adjustment. *Journal of Family Psychology*. 2008; 22:52–540.
- Gleason, JB. *The development of language*. Boston, MA: Pearson; 2005.

- Goodman SH, Brogan D, Lynch ME, Fielding B. Social and emotional competence in children of depressed mothers. *Child Development*. 1993; 64:516–531. [PubMed: 8477632]
- Goodman SH, Gotlib IH. Risk for psychopathology in the children of depressed mothers: A developmental model for understanding mechanisms of transmission. *Psychological Review*. 1999; 106(3):458–490. [PubMed: 10467895]
- Gross HE, Shaw DS, Moilanen KL, Dishion TJ, Wilson MN. Reciprocal models of child behavior and depressive symptoms in mothers and fathers in a sample of children at risk for early conduct problems. *Journal of Family Psychology*. 2008; 22:742–751. [PubMed: 18855510]
- Gross HE, Shaw DS, Burwell RA, Nagin DS. Transactional processes in child disruptive behavior and maternal depression: A longitudinal study from early childhood to adolescence. *Development and Psychopathology*. 2009; 21:139–156. [PubMed: 19144227]
- Hay, DF. Postpartum depression and cognitive development. In: Murray, L.; Cooper, PJ., editors. *Postpartum depression and child development*. New York: Guilford Press; 1997.
- Hyler SE. PDQ-4+ manual. New York State Psychiatric Institute. 1994
- Jöreskog, KG.; Sörbom, D. LISREL 8: User's reference guide. Lincolnwood, IL: SSI; 1996.
- Kane P, Garber J. The relations among depression in fathers, children's psychopathology and father-child conflict: A meta-analysis. *Clinical Psychology Review*. 2004; 24:339–360. [PubMed: 15245835]
- Klein DN, Lewinsohn PM, Rohde P, Seeley JR, Olino TM. Psychopathology in the adolescent and young adult offspring of a community sample of mothers and fathers with major depression. *Psychological Medicine*. 2005:353–365. [PubMed: 15841871]
- Knopik VS. Maternal smoking during pregnancy and child outcomes: Real or spurious effect? *Developmental Neuropsychology*. 2009; 34:1–36. [PubMed: 19142764]
- Kopp CB. Regulation of distress and negative emotions: A developmental view. *Developmental Psychology*. 1989; 25:343–354.
- Laflamme D, Pomerleau A, Malcuit G. A comparison of fathers' and mothers' involvement in childcare and stimulation behaviors during free-play with their infants at 9 and 15 months. *Sex Roles*. 2002; 47:507–518.
- Lamb, ME. The role of father in child development. 3. Hoboken, N.J.: John Wiley & Sons, Inc; 1997.
- Lamb, ME.; Oppenheim, D. Fatherhood and father-child relationships: Five years of research. In: Cath, SH.; Gurwitt, A.; Gunsberg, L., editors. *Fathers and their families*. Hillsdale, NJ: Analytic Press, Inc; 1989.
- Lesch KP. Gene-environment interaction and the genetics of depression. *Journal of Psychiatry & Neuroscience*. 2004; 29:174–184. [PubMed: 15173894]
- Leve LD, Kerr DC, Shaw D, Ge X, Neiderhiser JM, Scaramella LV, Reid JB, Conger R, Reiss D. Infant pathways to externalizing behavior: Evidence of genotype x environment interaction. *Child Development*. 2010:340–356. [PubMed: 20331671]
- Leve LD, Neiderhiser JM, Scaramella LV, Reiss D. The Early Growth and Development Study: Using the prospective adoption design to examine genotype-environment interplay. *Acta Psychologica Sinica*. 2008; 40:1106–1115. [PubMed: 19458782]
- Leve LD, Neiderhiser JM, Ge X, Scaramella LV, Conger RD, Reid JB, Shaw DS, Reiss D. The Early Growth and Development Study: A prospective adoption design. *Twin Research and Human Genetics*. 2007; 10:84–95. [PubMed: 17539368]
- Lieb R, Isensee B, Höfler M, Pfister H, Wittchen H. Parental major depression and the risk of depression and other mental disorders in offspring: A prospective-longitudinal community study. *Archives of General Psychiatry*. 2002; 59:365–374. [PubMed: 11926937]
- Loeber R, Green SM, Lahey BB, Strouthamer-Loeber M. Optimal informants on childhood disruptive behaviors. *Development and Psychopathology*. 1989; 1:317–337.
- Masten AS, Roisman GI, Long JD, Burt KB, Obradovic J, Riley JR, et al. Developmental cascades: Linking academic achievement, externalizing and internalizing symptoms over 20 years. *Developmental Psychology*. 2005; 41:733–746. [PubMed: 16173871]
- McBride BA, Mills G. A comparison of mother and father involvement with their preschool age children. *Early Childhood Research Quarterly*. 1993; 8:457–477.

- McNeil TF, Cantor-Graae E, Sjostrom K. Obstretic complications as antecedents of schizophrenia: Empirical effects of using different obstretic complication scales. *Journal of Psychiatric Research*. 1994; 28:519–530. [PubMed: 7699611]
- Mezulis AH, Hyde JS, Clark R. Father involvement moderates the effect of maternal depression during a child's infancy on child behavior problems in kindergarten. *Journal of Family Psychology*. 2004; 18:575–588. [PubMed: 15598163]
- Molenaar P. THEIL: A FORTRAN program for robust covariance matrix estimation. 1996 Unpublished program manual.
- Natsuaki MN, Ge X, Neiderhiser JM, Reiss D. Genetic liability, environment, and the development of fussiness in toddlers: The roles of maternal depression and parental responsiveness. *Developmental Psychology*. in press.
- Nicodemus KK, Marenco S, Batten AJ, Vakkalanka R, Egan MF, Straub R, et al. Serious obstretic complications interact with hypoxia-regulated/vascular-expression genes to influence schizophrenia risk. *Molecular Psychiatry*. 2008; 13:873–877. [PubMed: 18195713]
- Perez, SM.; Gauvain, M. The sociocultural context of transitions in early socioemotional development. In: Brownell, CA.; Kopp, CB., editors. *Socioemotional development in the toddler years: Transitions and transformations*. New York: Guilford; 2007.
- Porges SW, Doussard-Roosevelt JA, Portales LA, Suess PE. Cardiac vagal tone: Stability and relation to difficultness in infants and 3-year-olds. *Developmental Psychobiology*. 1994; 27:289–300. [PubMed: 7926281]
- Orvaschel H, Walsh-Allis G, Ye W. Psychopathology in children of parents with recurrent depressive. *Journal of Abnormal Child Psychology*. 1988; 16:17–28. [PubMed: 3361028]
- Oyserman D, Mowbray CT, Meares PA, Firminger KB. Parenting among mothers with a serious mental illness. *American Journal of Orthopsychiatry*. 2000; 70:296–315. [PubMed: 10953777]
- Radke-Yarrow, M. *Children of depressed mothers: From early childhood to maturity*. New York: Cambridge U. Press; 1998.
- Reyno SM, McGrath PJ. Predictors of parent training efficacy for child externalizing behavior problem - a meta-analytic review. *Journal of Child Psychology and Psychiatry*. 2006; 47:99–111. [PubMed: 16405646]
- Rice F, Harold GT, Thaper A. Assessing the effects of age, sex and shared environment on the genetic etiology of depression in childhood and adolescence. *Journal of Child Psychology and Psychiatry*. 2002; 43:1039–1051. [PubMed: 12455925]
- Robinson M, Oddy WH, Li J, Kendall GE, de Klerk NH, Silburn SR, et al. Pre- and postnatal influences on preschool mental health: A large-scale cohort study. *Journal of Child Psychology and Psychiatry*. 2008; 49:1118–1128. [PubMed: 19017026]
- Rook KS, Pietromonaco PR, Lewis MA. When are dysphoric individuals distressing to others and vice versa? Effects of friendship, similarity, and interaction task. *Journal of Personality and Social Psychology*. 1994; 67:548–559. [PubMed: 7965604]
- Rowe ML, Pan BA, Ayoub C. Predictors of variation in maternal talk to children: A longitudinal study of low-income families. *Parenting: Science and Practice*. 2005; 5:285–310.
- Rutter, M. Resilience reconsidered: Conceptual considerations, empirical findings, and policy implications. In: Shonkoff, JP.; Meisels, SJ., editors. *Handbook of early childhood intervention*. 2. New York: Cambridge U. Press; 2000.
- Shaw DS, Bell RQ, Gilliom M. A truly early starter model of antisocial behavior revisited. *Clinical Child and Family Psychology Review*. 2000; 3:15–172.
- Shaw DS, Dishion TJ, Wilson MN, Gardner F. Improvements in maternal depression as a mediator of intervention effects on early child problem behavior. *Development and Psychopathology*. 2009; 21:417–439. [PubMed: 19338691]
- Shaw DS, Gilliom J, Ingoldsby EM, Nagin D. Trajectories leading to school-age conduct problems. *Developmental Psychology*. 2003; 39:189–200. [PubMed: 12661881]
- Shaw DS, Schonberg M, Sherrill J, Huffman D, Lukon J, Obrosky D, et al. Responsivity to Offspring's Expression of Emotion Among Childhood-Onset Depressed Mothers. *Journal of Clinical Child and Adolescent Psychology*. 2006; 35(4):490–503. [PubMed: 17007595]

- Stansbury K, Gunnar MR. Adrenocortical activity and emotion regulation. *Monographs of the Society for Research in Child Development*. 1994; 59:250–283.
- Sullivan PF, Neale MC, Kendler KS. Genetic epidemiology of major depression: Review and meta-analysis. *American Journal of Psychiatry*. 2000; 157:1552–1562. [PubMed: 11007705]
- Tannenbaum L, Forehand R. Maternal depressive mood: The role of the father in preventing adolescent problem behaviors. *Behaviour Research and Therapy*. 1994; 32:321–325. [PubMed: 8192631]
- Teti DM, Gelfand DM. Behavioral competence among mothers of infants in the first year: The mediational role of maternal self-efficacy. *Child Development*. 1991; 62:918–929. [PubMed: 1756667]
- Tiedje LB, Darling-Fisher CS. Factors that influence fathers' participation in child care. *Health Care for Women International*. 1993; 14:99–107. [PubMed: 8454530]
- Tremblay, RE.; Nagin, DS. The developmental origins of physical aggression in humans. In: Tremblay, RE.; Hartup, WH.; Archer, J., editors. *Developmental origins of aggression*. New York: Guilford Press; 2005. p. 83-106.
- Tully EC, Iacono WG, McGue M. An adoption study of parental depression as an environmental liability for adolescent depression and childhood disruptive disorders. *The American Journal of Psychiatry*. 2008; 165:1148–1154. [PubMed: 18558644]
- Üstün TB, Ayuso-Mateos JL, Chatterji S, Mathers C, Murray CJL. Global burden of depressive disorders in the year 2000. *British Journal of Psychiatry*. 2004; 184:386–392. [PubMed: 15123501]
- Wakschlag LS, Leventhal BL, Pine DS, Pickett KE, Carter AS. Elucidating early mechanisms of developmental psychopathology: The case of prenatal smoking and disruptive behavior. *Child Development*. 2006; 77:893–906. [PubMed: 16942496]
- Weissman MM, Gammon GD, John K, Merikangas KR, Warner V, Prusoff BA, D, et al. Children of depressed parents: Increased psychopathology and early onset of major depression. *Archives of General Psychiatry*. 1987; 44:847–853. [PubMed: 3662741]
- Weissman MM, Pilowsky DJ, Wickramaratne PJ, Talati A, Wisniewski SR, Fava, et al. Remissions in maternal depression and child psychopathology: A STAR*D-Child report. *JAMA: Journal of the American Medical Association*. 2006; 295:1389–1398.
- Weissman MM, Wolk S, Wickramaratne P, Goldstein RB, Adams P, Greenwald S, et al. Children with prepubertal-onset major depressive disorder and anxiety grown up. *Archives of General Psychiatry*. 1999; 56:794–801. [PubMed: 12884885]
- Welner Z, Reich W, Herjanic B, Jung K, Amado H. Reliability, validity and parent-child agreement studies of the Diagnostic Interview for Children and Adolescents (DICA). *Journal AM Acad Child Adolesc Psychiatry*. 1987; 236:649–653. [PubMed: 3667494]
- Wille DE. The 1990s: Gender differences in parenting roles. *Sex Roles*. 1995; 33:803–817.

Figure 1.

Example structural equation model where the timing of the influence of AP depressive symptoms on child externalizing problems at 27 months is tested in the presence of BM depressive symptoms. Each AP measurement has a direct path to child externalizing problems.

Figure 2.

Example structural equation model where a latent factor of AP depressive symptoms predicts child externalizing problems at 27 months in the presence of BM depressive symptoms. Each AP measurement is loaded onto a latent factor of AP depressive symptoms.

Figure 3.

Structural equation model predicting child externalizing problems at 27 months. This full model includes, AFs, AMs, BM, pregnancy risk, and 9 month child fussiness. Solid lines represent significant paths; dotted lines are insignificant.

Note. + = $p < .10$; * = $p < .05$.

Figure 4.

Structural equation model predicting child externalizing problems at 27 months. This full model includes, AFs, AMs, BM, pregnancy risk, and 9 month child fussiness. This model includes the indirect effect of BM depressive symptoms on 27 child externalizing symptoms through pregnancy risk and the effect of AF 9 month depressive symptoms on AM depressive symptoms.

Note. + = $p < .10$; * = $p < .05$.

Table 1

Demographics for Birth Parents and Adoptive Parents

Variable	BM	BF	AM	AF
Mean age (years)	24.16 +/- 5.93	25.41 +/- 7.34	37.99 +/- 5.48	38.72 +/- 5.89
Race (%)				
Caucasian	77	63	93	92
African-American	11	20	4	5
Hispanic/Latino	4	8	1	1
Multiethnic	5	5	2	2
Other ^a	2	4	1	1
Mean education level	5	5	9	9
Median annual house income	\$14,000	\$21,000	\$100,000	
Mean number of individuals in home	3.6	3.5	3.6	

Note. BM = birth mother; BF = birth father; AM = adoptive mother; AF = adoptive father.

Education scores were as follows: 1 (< 8th grade), 2 (completed 8th grade), 3 (completed 12th grade), 4 (some trade school), 5 (completed trade school), 6 (some junior college), 7 (completed junior college), 8 (some college), 9 (completed college), 10 (some graduate/professional school), and 11 (completed graduate/professional school).

^a Includes Asian, Native American/Pacific Islander, American Indian/Alaskan Native, and unknown.

Table 2

Descriptive Statistics

Measure (N) Child age	Mean	SD	Min	Max
BM Depressive Symptoms (N=346) Pregnancy History Calendar (reduced items)	9.16	4.60	5	40
BM Depressive Symptoms (N=344) 4 months	11.18	9.78	0	54
BM Depressive Symptoms (N=312) 18 months	11.23	9.37	0	43
BF Depressive Symptoms (N=102) 4 months	6.96	6.92	0	23
BF Depressive Symptoms (N=95) 18 months	7.68	7.17	0	35
AM Depressive Symptoms (N=346) 9 months	3.59	3.15	0	15
AM Depressive Symptoms (N=336) 18 months	3.75	3.85	0	25
AM Depressive Symptoms (N=328) 27 months	3.87	4.30	0	30
AF Depressive Symptoms (N=335) 9 months	2.87	3.38	0	27
AF Depressive Symptoms (N=310) 18 months	2.82	3.39	0	22
AF Depressive Symptoms (N=312) 27 months	2.69	3.50	0	22
Child Externalizing Symptoms (AM report; N=304) 27 months	11.49	6.14	0	27
Child Externalizing Symptoms (AF report; N=289) 27 months	10.35	6.35	0	36
Child Externalizing Symptoms (Worst Score; N=309) 27 months	13.46	5.98	0	36

Note. BM = birth mother; BF = birth father; AM = adoptive mother; AF = adoptive father.

Table 3

Results table of structural equation models

Model	RMSEA ^a	CFI ^b	χ^2 Significance ^c	AIC ^d	Significant paths predicting child CBCL at 27 mo
AM and BM Depressive Symptoms and Toddler Externalizing Problems					
Full with all covariates	0.054	0.88	$p < .05$	383.58	<ul style="list-style-type: none"> • BM ($p < .10$) • 9 mo fussiness ($p < .05$)
Trimmed covariates & AM as latent factor	0.10	0.77	$p < .05$	288.22	<ul style="list-style-type: none"> • AM latent ($p < .05$) • BM ($p < .05$) • 9 mo fussiness ($p < .05$)
AF and BM Depressive Symptoms and Toddler Externalizing Problems					
Full with all covariates	0.055	0.88	$p < .05$	391.35	<ul style="list-style-type: none"> • AF Wave A ($p < .10$) • BM ($p < .05$) • 9 mo fussiness ($p < .05$) • Pregnancy risk ($p < .10$)
Trimmed covariates & AM as latent factor	0.055	0.94	$p < .05$	186.51	<ul style="list-style-type: none"> • AF Wave A ($p < .05$) • 9 mo fussiness ($p < .05$) • Pregnancy risk ($p < .10$)
AM, AF, and BM Depressive Symptoms and Toddler Externalizing Problems					
Full Model	0.043	0.95	$p < .05$	299.02	<ul style="list-style-type: none"> • AM latent ($p < .05$) • AF Wave A ($p < .05$) • BM ($p < .10$) • 9 mo fussiness ($p < .05$)
Indirect genetic effect through pregnancy risk	0.043	0.95	$p < .05$	296.33	<ul style="list-style-type: none"> • AM latent ($p < .05$) • AF Wave A ($p < .05$) • 9 mo fussiness ($p < .05$) • BM to pregnancy risk ($p < .05$) • Pregnancy risk ($p < .05$)

Model	RMSEA ^a	CFI ^b	χ^2 Significance ^c	AIC ^d	Significant paths predicting child CBCL at 27 mo
Addition of path from AF Wave A to AM latent factor	0.042	0.96	$p < .05$	203.25	<ul style="list-style-type: none">• AM latent ($p < .05$)• AF Wave A ($p < .05$)• 9 mo fussiness ($p < .05$)• BM to pregnancy risk ($p < .05$)• Pregnancy risk ($p < .05$)• AF Wave A to AM latent ($p < .05$)

Note.

^aFor RMSEA, a value less than .05 is considered a good fit, and below .08 an adequate fit;

^bThe CFI should be equal or greater than .90 to accept the model;

^cChi-square is influenced by sample size and indicates good fit when $p > .05$. AIC was used to compare models;

^dA lower AIC indicates a better fit.