

# Influences of Biological and Adoptive Mothers' Depression and Antisocial Behavior on Adoptees' Early Behavior Trajectories

David C. R. Kerr · Leslie D. Leve · Gordon T. Harold ·  
Misaki N. Natsuaki · Jenae M. Neiderhiser ·  
Daniel S. Shaw · David Reiss

Published online: 14 February 2013  
© Springer Science+Business Media New York 2013

**Abstract** Research clearly demonstrates that parents pass risk for depression and antisocial behavior on to their children. However, most research confounds genetic and environmental mechanisms by studying genetically related individuals. Furthermore, most studies focus on either depression or antisocial behavior in parents or children, despite evidence of co-occurrence and shared etiology, and few consider the early origins of these problems in childhood. We estimated the influence of biological and adoptive mothers' depression and antisocial behavior on growth in child externalizing and internalizing behaviors across early childhood using data from a prospective adoption study. Participants were 346 matched triads of physically healthy children (196 boys; 150 girls), biological mothers (BM), and adoptive mothers (AM). Latent

growth curve models were estimated using AM reports of child internalizing and externalizing behaviors at ages 18, 27, and 54 months. Predictors of intercept (18 months) but not slope were identified. BM lifetime histories of major depressive disorder predicted child externalizing behaviors and BM antisocial behavior predicted child internalizing behavior. AM depressive symptoms and antisocial behavior were associated with both child outcomes. AM paths, but not BM paths were partially replicated using adopted fathers' reports of child outcomes. BM obstetric complications, prenatal depressive symptoms, and postnatal adoptive family contact with BM did not account for BM paths. This adoption study distinguished risks conferred by biological mothers' depression and antisocial behavior to children's behaviors from those associated with adoptive mothers' related symptoms. Future studies should examine gene-environment interplay to explain the emergence of serious problem trajectories in later childhood.

D. C. R. Kerr (✉)  
School of Psychological Science, Oregon State University,  
Reed Lodge, Corvallis, OR 97331, USA  
e-mail: david.kerr@oregonstate.edu

D. C. R. Kerr · L. D. Leve  
Oregon Social Learning Center,  
10 Shelton McMurfhey Boulevard,  
Eugene, OR 97401, USA

G. T. Harold  
University of Leicester, Leicester, UK

M. N. Natsuaki  
University of California, Riverside, Riverside, CA, USA

J. M. Neiderhiser  
The Pennsylvania State University, University Park, PA, USA

D. S. Shaw  
University of Pittsburgh, Pittsburgh, PA, USA

D. Reiss  
Yale Child Study Center, Yale School of Medicine,  
New Haven, CT, USA

**Keywords** Depression · Externalizing · Internalizing · Early childhood · Genetic risk · Adoption

Depression and antisocial behavior problems are significant clinical, social, and economic concerns (Greenberg et al. 2003; Welsh, et al. 2008). These problems often co-occur, but are increasingly distinguishable over the course of development (Angold and Costello 1993; Kendler et al. 2003; Krueger and Markon 2006). Although distinct externalizing (e.g., aggression and oppositionality) and internalizing (e.g., anxiety, sadness) factors are difficult to discern during toddlerhood (Gjone and Stevenson 1997; van den Oord et al. 2000), the problems have different sequelae and demand different preventative measures. Thus, identifying the early origins and predictors of these behaviors is critical to aid the development of programs to prevent later problems.

Multigenerational and epidemiological research highlights how parents' depression and antisocial behavior influence early childhood problems (Conger et al. 2009; Olino et al. 2008). For example, the odds of a child developing depression by age 16 years are nearly five times greater if his/her mother was depressed than if she was not (Murray et al. 2011) and a child with an affectively-ill parent has a 40 % chance of being diagnosed with major depressive disorder (MDD) by age 20 (Beardslee et al. 1998). Prospective multigenerational studies also support that the externalizing behavior parents showed in their childhood is associated with that of their children (Conger et al. 2009). However, such studies have almost always been based on genetically-related parent-child dyads, and therefore fully confound genetic and environmental influences.

Several genetically-informed designs can better distinguish these influences and have contributed to the understanding of why depression and antisocial behavior show intergenerational continuity. These studies are especially important because they suggest mechanisms by which risks for these problems are transmitted from parents to children, and therefore pathways through which intervention efforts might be most successful. For example, an adoption study reported that risk for depression among children living with a depressed mother was elevated among genetically-related (nonadopted) and unrelated (adopted) children, suggesting underlying environmental rather than genetic mechanisms (Tully et al. 2008). Two other studies also support the influence of rearing mothers' depression on children's functioning. Among mothers who used in vitro fertilization, correlations between mothers' and children's internalizing symptoms were no stronger for genetically-related than for unrelated pairs (Lewis et al. 2011). Further, a children of twins study highlighted direct environmental pathways of depression risk transmission, and both genetic and environmental pathways from parent depression to child conduct problems (Silberg et al. 2010). Extended twin and in vitro studies also elucidate how parental antisocial behavior impacts children's externalizing and more serious antisocial behavior. These studies support shared family influences serving as a mechanism of intergenerational transmission (Meyer et al. 2000), and direct associations between parent antisocial behavior and child antisocial behavior in genetically-unrelated (and related) families (Harold et al. 2011). Twin studies also have some relevance to questions of intergenerational continuity insofar as they support both genetic and environmental influences on depression and antisocial behavior (e.g., Hicks et al. 2004; Sullivan et al. 2000; van der Valk et al. 2003). Despite this growing literature, few studies have examined the early childhood manifestations of intergenerational problem transmission.

In one exception, adoptive mothers' depressive symptoms when children were ages 9 to 27 months, and biological mothers' depressive symptoms (prenatal and postpartum) predicted children's externalizing behaviors at age 27 months (Pemberton et al. 2010). While this study provided important evidence

regarding the early manifestations of risk conferred by adoptive and biological mothers' depressive symptoms, several critical issues remain unclear. First, it is unknown whether maternal symptoms are associated with absolute levels of child behaviors at 27 months only or with earlier problems and change over time. Second, there was some indirect evidence that higher rates of obstetric complications among biological mothers who reported depressive symptoms may explain some of their influence on child externalizing behaviors. This point requires explicit testing, as well as consideration with respect to child internalizing behaviors. Third, it remains unclear whether the effects of (biological or adoptive) maternal antisocial behavior can be distinguished from the effects of depressive symptoms on child behaviors during early childhood; and likewise whether such transmission pathways to child externalizing outcomes are distinct from those shared with internalizing behaviors. Given the prevalence of and genetic basis for co-occurring symptomatology (e.g., O'Connor et al. 1998), overlap and cross-symptom prediction seem likely. Indeed, several studies indicate that maternal antisocial behavior partially accounts for relations between maternal depression and child antisocial behavior in early childhood (Kim-Cohen et al. 2005). Others have found maternal internalizing symptoms to be associated with both child externalizing and internalizing symptoms (Kim et al. 2009).

Thus, the present study uses the same adoption sample as Pemberton et al. (2010) to address gaps in the field by examining: (1) predictors of child outcomes from ages 18–54 months; (2) externalizing and internalizing spectrum behaviors in both children and (biological and adoptive) mothers; (3) genetic effects of biological mothers' depression diagnosis, which may index more significant and trait-level risk than the symptom counts across shorter time frames used in Pemberton et al. (2010); and (4) genetic effects of biological mothers' antisocial behaviors.

Of note, the adoption design does not wholly rule out some competing explanations for apparent genetic pathways of influence from biological mothers' symptoms to child outcomes. Namely, biological mothers with histories of depression and antisocial behavior may be more likely to expose offspring to adverse prenatal environments associated with child behavior problems or early risks for such problems (e.g., Beck and Shaw 2004; Brennan et al. 2008). Additionally, given that modern adoption arrangements often include some contact among children and their biological and adoptive families, it remains possible that biological mothers' symptoms and behaviors may impact their children via environmental mechanisms. Thus, we examined three competing explanations for ostensibly genetic pathways of influence in study models; namely, obstetric complications, prenatal depression, and adoption openness/contact between biological and adoptive families.

In the present study, we estimated levels and changes over time in early externalizing and internalizing behaviors in adopted children. We expected normative decreases in

externalizing behaviors and increases in internalizing behaviors across this early childhood developmental period (Gilliom and Shaw 2004). We then considered adoptive and biological mothers' depression/depressive symptoms and antisocial behavior as indices of environmental and genetic risk, respectively, for child externalizing and internalizing behaviors. We expected that risks transmitted by mothers (biological and adoptive) would be conferred in toddlerhood or manifested over time as deviations from developmentally-expected changes in child behaviors.

First, we hypothesized that biological mothers' major depressive disorder (MDD) histories and/or adult antisocial behavior each would be associated with children's higher levels of internalizing and externalizing behaviors at 18 months (i.e., intercept), as well as less steeply decreasing levels of externalizing, and more steeply increasing levels of internalizing behaviors (i.e., more positive slope) across early childhood. Slope predictions were based on the expectations that: (1) risks for externalizing problems may be more evident once normative developmental struggles with self-regulation that underlie the peak prevalence in early childhood have resolved for most children; and (2) risks for internalizing problems may become apparent when children develop the capacities to anticipate the future and clearly convey internal states of distress (Gilliom and Shaw 2004).

Second, we expected adoptive mothers' depressive symptoms (lifetime diagnoses were not ascertained and were considered less relevant than symptoms during the child's life) and/or antisocial behavior each to be associated with higher levels of children's externalizing and internalizing behaviors at 18 months. These hypotheses are based on research showing that maternal psychopathology negatively impacts child behaviors by compromising the quality of parenting (Conger et al. 2009). However, few studies have examined these processes in genetically-unrelated families, which eliminates the possible effects of shared genes between parents and children in biological families. We expected that some environmental conditions associated with parental psychopathology may not emerge by toddlerhood, or may be evoked by later child behaviors (Scaramella and Leve 2004). Thus, we further hypothesized that adoptive mothers' psychopathology would predict higher behavior slopes to age 54 months.

Third, we tested whether any apparent genetic effects would be explained by negative prenatal conditions or biological mothers' prenatal depressive symptoms. A moderation model also was tested to determine whether there would be stronger evidence of problem transmission between biological mothers and children when there was greater contact between the adoptive and biological families; such a finding would be suggestive of environmental rather than wholly genetic effects of biological mothers' problems on child outcomes.

Finally, we explored whether risks conferred by (biological and adoptive) maternal depression and antisocial behavior

would be specific to child externalizing or internalizing behaviors. Very little is known about the specificity of genetic and environmental transmission of risk for depression and antisocial behavior during early childhood, despite evidence for distinguishable patterns in adult samples (Hicks et al. 2004; Kendler et al. 2003). Therefore, this hypothesis was exploratory rather than directional. In all, the prospective adoption design permitted us to break new ground with respect to understanding parent-child problem transmission pathways for related and unrelated dyads.

## Method

### Participants

Participants were drawn from the first cohort ( $n=361$ ) of the Early Growth and Development Study, a multisite longitudinal study of physically healthy (e.g., no known major medical conditions or surgeries, and children were born no more than 3 weeks premature), domestically adopted U.S. children and their birth and adoptive parents (Leve et al. 2013). Children from a range of racial backgrounds (58.4 % White) participated. Adoptive parents<sup>1</sup> were predominantly Caucasian (92 %), educated (mean level="completed college"), middle-class (median annual household income=\$100,000), and in their late 30s [mean (SD) ages of adoptive mothers and fathers=37.99 (5.48), and 38.72 (5.89), respectively]. Birth mothers and fathers were primarily Caucasian (77 % and 63 %, respectively) or African American (11 % and 20 %, respectively), less educated (mean="completed trade school"), lower SES (median annual house income=\$14,000 and \$21,000, respectively), and younger (mean (SD) ages=24.16 (5.93) and 25.41 (7.31), respectively).

Children were placed with nonrelative adoptive parents within 3 months of birth ( $M=7.11$  days postpartum,  $SD=13.28$ ). All study procedures were approved by institutional review boards of study sites. After complete description of the study to adult participants, written informed consent (assent and parental consent for biological parents who were minors) was obtained. Families were paid for participation. Cases were selected for these analyses if one or more assessments of the child outcome and an assessment of either biological or adoptive mothers' depression or antisocial behavior were available ( $n=346$ ; 96 % of the total cohort; 196 males, 150 females).

<sup>1</sup> A subset ( $n=13$ , 3.7 %) of adoptive families included a divorced single father or two adoptive fathers. In these families, the divorced father or the father from the same-sex couple deemed the primary caregiver completed the measures labeled as "adoptive mother." As models that included or excluded these families were not significantly or substantively different, all families were included in present models. Space precluded repeatedly qualifying the use of the label "adoptive mothers."

## Measures

*Adoptive Mothers' Depressive Symptoms* Mothers completed the self-reported Beck Depression Inventory (BDI; Beck and Steer 1993) when children were age 18 months ( $\alpha=0.80$ ). Participants chose one of four statements ranging from neutral (0) to depressed feelings (1–3) about life in the past week. The suicidal ideation item was not administered in the present study. Thus, mothers completed 20 items from this 21-item scale, and total scores could range from 0 to 60. Raw scores were used in analyses; prorated scores were used in comparisons to the BDI scores suggestive of mild, moderate, and severe depression (10–18, 19–29, and 30–63, respectively; Beck et al. 1988).

*Adoptive Mothers' Antisocial Behavior* Mothers completed an adaptation of the Antisocial Action Scale (Levenson et al. 1995) when children were age 18 months. Mothers' self-reports on 13 items indicative of dishonest and antisocial behaviors (e.g., lying, stealing, not helping others) were summed ( $\alpha=0.56$ ) using a 4-point Likert scale ranging from 1 (*never*) to 4 (*often*); therefore, total scores could range from 13 to 52.

*Biological Mothers' Lifetime History of MDD* At 18 months postpartum, mothers completed the Composite International Diagnostic Interview (Kessler and Üstün 2004), a standardized interview created for nonclinical staff to assess MDD, among other conditions, based on DSM-IV (American Psychiatric Association 1994). Interviewers achieved at least 80 % agreement with supervisors on ratings of interviews with pilot participants before assessing study participants, and then maintained at least 80 % agreement with supervisors on the first 2 interviews and a subset (15 %) of subsequent interviews. Test-retest reliabilities reported in the literature for each disorder range from  $\kappa=0.45$  to 0.63. MDD history was coded (no=0, yes=1).

*Biological Mothers' Adult Antisocial Behaviors* Mothers completed the antisocial personality disorder (ASPD) module of the Computerized-Diagnostic Interview Schedule (Blouin et al. 1988) at 18 months postpartum. Others have found test-retest reliability for the ASPD diagnosis acceptable (e.g.,  $\kappa=0.527$ , Cottler et al. 1998). Twenty-two mothers met criteria for ASPD (adult antisocial behavior and a history of conduct disorder [CD]), 116 met criteria for adult antisocial behavior but not a CD history, and 167 did not meet adult antisocial behavior criteria. The former two groups were collapsed (no=0, yes=1) given that offspring of parents with adult antisocial behavior only or adult antisocial behavior plus childhood conduct disorder have shown equally higher rates of conduct disorder than offspring of parents with no antisocial diagnoses or conduct disorder

only (Elkins et al. 1997); this two-category variable formed an index of adult antisocial behavior.

*Biological Mothers' Prenatal Depressive Symptoms* When children were age 4 months, biological mothers reported on their depressive symptoms during pregnancy. Mothers who endorsed either sadness or anhedonia for at least a 2-week period during pregnancy were asked 5 items from the BDI, adapted to the 9-month reporting timeframe. Total scores were a sum of the sadness and anhedonia screening items (each scored no=0, yes=2) and the five BDI items (each on scale from 0 to 3); thus total scores could range from 0 to 19. For descriptive purposes only, scores were prorated to permit approximate comparison with the BDI clinical cut-off scores described above. Given its non-normal distribution and zero-inflation, the variable was dichotomized according to the presence (1) or absence (0) of significant symptoms as indicated by the endorsement of either screening item.

*Obstetric Complications* When children were age 4 months, biological mothers reported on complications during pregnancy (e.g., illness), labor and delivery (e.g., cord complications), and the neonatal period (e.g., prematurity) using a pregnancy screener and calendar method. Scoring was derived from the McNeil and Sjöström Scale for Obstetric Complications, weighted for severity (McNeil and Sjöström 1995).

*Child Externalizing And Internalizing Behavior* Adoptive mothers and fathers independently completed the Child Behavior Checklist for ages 1½-5 years (Achenbach and Rescorla 2000) when children were ages 18, 27, and 54 months. This instrument consists of 99 behaviors rated 0 (not true), 1 (sometimes true), or 2 (very true) over the last 2 months. Syndrome scores correspond to externalizing and internalizing behaviors. Prior research indicates comparable factor structure for externalizing and internalizing spectrum behaviors from ages 18 to 30 months, as well as stronger temporal stability within than across these spectra (Mathiesen and Sanson 2000); associations also have been reported between children's problems on the CBCL at 24 months with those at 6 years, which further supports predictive validity (Shaw et al. 2000). Raw scores were constructed identically across time ( $\alpha=0.71-0.92$ ). Mother reports (available for  $n=328$ , 312, and 253 at ages 18, 27, and 54 months, respectively) were used unless otherwise stated.

*Adoption Openness* Contact and shared knowledge between birth and adoptive families could overestimate genetic or environmental influences. Therefore, we considered adoptive parents' and birth mothers' ratings of the perceived openness of the relationship months as a possible moderator of paths of biological mothers' influence on child outcomes. Informants rated overall openness of the adoption process and relationship



using a 7-point scale (1=very closed, 7=very open). A prior study (Pemberton et al. 2010) with this sample found significant variability in the degree of openness according to birth mothers and adoptive mothers and fathers, but on average relatively open arrangements (openness means=4.6 to 5.1). A high rate of convergence was found among informants on openness ratings, as evidenced by standardized latent factor loadings ranging from 0.73 to 0.90. Informant scales were averaged to create an aggregate variable.

Missing Data

Forty-two (12 %) and 51 (15 %) cases were missing biological mothers’ MDD or adult antisocial behavior histories, respectively; both types of data were missing for 35 (10 %) cases. Thirteen cases (4 %) and nine cases (3 %) were missing adoptive mothers’ depressive symptoms and antisocial behaviors, respectively. Of the 346 children assessed up to three times, 219 (63 %) had outcome data at all three time points, 109 (32 %) at two time points, and 18 (5 %) at one time point. Twelve (4 %) of the 346 children in the analytic sample had no adoptive father reports; 196 (57 %), 110 (32 %), and 28 (8 %) were assessed by adoptive father report at three, two, or only one time point, respectively. Data that are missing as a function of observed covariates and outcomes are accommodated by maximum likelihood estimation in Mplus version 6.1 (Muthén and Muthén 1998–2010).

Data Analysis

Latent growth modeling was used to test a sequence of models run separately by child outcome. In all analyses, two-tailed tests were used, and a *p*-value of less than 0.05 was used as the criterion for judging statistical significance; model fit was judged based on a  $\chi^2$  with a *p*-value greater than 0.05, RMSEA close to 0 (less than 0.08) and CFI and TLI close to 1. In Step 1, we fit models that described how child behaviors changed over time. In Step 2, growth factors were regressed on biological and adoptive mothers’ depression and antisocial behavior separately (univariate) and then simultaneously (multivariate); whether findings replicated using adoptive fathers’ reports of child outcomes was then tested. In Step 3, we examined whether relations observed at Step 2 between biological mothers’ depression and antisocial behavior and child growth factors would persist when controlling for obstetric complications or biological mothers’ prenatal depressive symptoms; we also tested whether pathways from biological mothers’ psychopathology measure to child growth factors were moderated by adoption openness (i.e., were these interactions significant). Finally, we used parallel process growth models to explore whether predictive paths established in Step 2 were specific to child outcome.

Results

Descriptive Statistics

Information on study predictors and covariates is reported in Table 1, with child behavior outcomes in Table 2. Not surprisingly, adoptive mothers showed relatively low levels of depressive symptoms and antisocial behavior on average. Few adoptive mothers reported clinically significant depressive symptoms; based on prorated scores, 10.5 % (*n*=35) had scores above 9 (i.e., at least mild depression), and 1 % (*n*=3) had scores above 18 (i.e., at least moderate depression). Their antisocial behavior scores also were low (range of 13–28, within the possible range of 13–52), with the mean (16.8) close to the lowest possible score, and indicative of an endorsement of “rarely” on two of the 13 items. Biological mothers showed relatively high lifetime rates of MDD (29 %) and antisocial behavior (45 %). Regarding biological mothers’ prenatal depression, 38 % denied both screening items for significant symptoms; based on prorated scores, approximately 16 %, 11 %, 20 %, and 15 % reported prenatal depressive symptoms in the minimal, mild, moderate, and severe ranges, respectively.

As expected for a nonclinical sample, most children’s behavior was in the normal range; at ages 18, 27, and 54 months, mean (SD; range) T-scores for adoptive mother-reported externalizing behaviors were 47.93 (8.00; 28–70), 48.48 (8.31; 28–66), and 50.07 (8.85; 28–82), respectively; mean (SD; range) T-scores for internalizing behaviors were 43.91 (7.70; 29–69), 45.48 (8.17; 29–69), and 48.89 (8.93; 29–77), respectively. Despite low rates of clinically significant symptoms at any given assessment wave for either externalizing or internalizing behaviors, 82 children (23.6 %) reached or exceeded the borderline clinical range and 26 (7.5 %) reached the clinical range on at least one behavior type in at least one wave. Given that the predictive validity of the CBCL scales in toddlerhood have not frequently been reported, we

**Table 1** Descriptive statistics for study predictors and controls

	Mean (SD) or % positive	Range
AM depressive symptoms ( <i>n</i> =333)	3.86 (3.86)	0–25
AM antisocial behavior ( <i>n</i> =337)	16.90 (2.47)	13–28
BM MDD lifetime history ( <i>n</i> =304)	29.6 %	0–1
BM adult antisocial behavior ( <i>n</i> =295)	45.4 %	0–1
BM prenatal depressive symptoms ( <i>n</i> =343)	62.1 %	0–1
Obstetric complications ( <i>n</i> =346)	10.33 (6.98)	0–42
Adoption openness ( <i>n</i> =346)	0.04 (0.94)	–2.06–1.84

AM Adoptive mother, BM Biological mother, MDD major depressive disorder history

note that externalizing behaviors at age 18 months were correlated with those at 27 and 54 months ( $r=0.65$  and  $0.45$ , respectively,  $p<0.001$ ), as were internalizing behaviors ( $r=0.58$  and  $0.39$ , respectively,  $p<0.001$ ), suggesting some stability of individual differences over time.

#### Step 1: Unconditional Growth Models

Growth models (Step 1) were run separately for externalizing and internalizing behavior outcomes; linear models fit best (see Table 2). Means and variances in intercepts and slopes were significantly different from zero; slope effects indicated increases in both behaviors across 18 to 54 months. Child gender did not predict the growth factors for either child outcome.

#### Step 2: Regression of Child Outcomes on Indices of Genetic and Environmental Influence

In univariate models, externalizing behavior intercepts were predicted by biological mothers' MDD,  $\beta=0.18$ ; adoptive mothers' depressive symptoms,  $\beta=0.21$ , and antisocial behavior,  $\beta=0.23$ , all  $p<0.05$ ; and marginally by biological mothers' antisocial behavior,  $\beta=0.12$ ,  $p=0.08$ . Internalizing behavior intercepts were predicted by biological mothers' antisocial behavior,  $\beta=0.15$ ; adoptive mothers' depressive symptoms,  $\beta=0.20$ ; adoptive

mothers' antisocial behavior,  $\beta=0.30$ , all  $p<0.05$ ; and marginally by biological mothers' MDD,  $\beta=0.12$ ,  $p=0.08$ . Predictors were not associated with child behavior slopes.

Multivariate regressions reported in Table 3 were similar to univariate patterns, with modest apparent diminutions in magnitudes of effect; for example, among the strongest was from  $\beta=0.20$  to  $\beta=0.13$ ,  $ps<0.05$ , for the association between adoptive mothers' depressive symptoms and child internalizing behavior intercept. Associations from biological mothers' antisocial behavior to child externalizing intercept, and from biological mothers' MDD to child internalizing intercept were similar in magnitude but no longer marginally significant. Thus, results of univariate versus multivariate models suggested little overlap in predictive pathways. Correlations between mothers' depressive symptoms and antisocial behavior indicated modest comorbidity or co-occurrence for both biological ( $r=0.17$ ) and adoptive mothers ( $r=0.29$ ) and yielded no concerns regarding multicollinearity. In the separate models by outcome, the four predictors accounted for significant variance in externalizing and internalizing intercepts,  $R^2=0.12$  and  $0.12$ , respectively,  $p<0.01$ , but not in slopes,  $R^2=0.03$  and  $0.04$ , respectively.

**Table 2** Unconditional linear growth curve models conducted separately by outcome and informant ( $n=346$  mother-reports and  $n=334$  father-reports)

	Mother-reports		Father-reports	
Child EXT behaviors model	Mean (range)	SD	Mean (range)	SD
Observed scores (growth weight <sup>a</sup> )				
EXT-18 months (0)	11.06 (0–31)	6.01	9.56 (0–30)	6.07
EXT-27 months (0.75)	11.55 (0–27)	6.09	10.35 (0–36)	6.33
EXT-54 months (3)	12.79 (0–39)	6.92	11.15 (0–35)	6.67
Latent growth factors	Mean (SE)	Variance (SE)	Mean (SE)	Variance (SE)
EXT-intercept	11.12 (0.31)***	25.27 (2.80)***	9.70 (0.32)***	25.55 (2.98)***
EXT-slope	0.58 (0.14)***	2.36 (1.15)*	0.48 (0.13)***	2.49 (1.18)*
Child INT behaviors model	Mean (range)	SD	Mean (range)	SD
Observed scores (growth weight <sup>a</sup> )				
INT-18 months (0)	5.01 (0–24)	3.47	4.54 (0–23)	3.88
INT-27 months (0.75)	5.80 (0–24)	4.10	5.25 (0–25)	4.36
INT-54 months (3)	7.72 (0–36)	5.44	6.59 (0–25)	5.25
Latent growth factors	Mean (SE)	Variance (SE)	Mean (SE)	Variance (SE)
INT-intercept	5.01 (0.18)***	8.30 (1.06)***	4.59 (0–23)	10.63 (1.29)***
INT-slope	0.90 (0.11)***	1.31 (0.61)*	0.68 (0.11)***	1.38 (0.66)*

<sup>a</sup> growth weights set to specify time in years. EXT=Child externalizing behaviors. INT=Child internalizing behaviors. SE=standard error. Sample sizes at ages 18, 27, and 54 months were  $n=328$ ,  $312$ , and  $253$ , respectively, for mother-reports;  $n=311$ ,  $296$ , and  $229$ , respectively, for father-reports. Mother-report EXT Model  $\chi^2$  ( $n=346$ ,  $df=1$ )= $0.11$ ,  $p=0.87$ ; CFI=1.000, TLI=1.011, RMSEA=0.000. Father-report EXT Model  $\chi^2$  ( $n=334$ ,  $df=1$ )= $1.37$ ,  $p=0.24$ ; CFI=0.998, TLI=0.995, RMSEA=0.033. Mother-report INT Model  $\chi^2$  ( $n=346$ ,  $df=1$ )= $0.51$ ,  $p=0.48$ ; CFI=1.000, TLI=1.008, RMSEA=0.000. Father-report INT Model  $\chi^2$  ( $n=334$ ,  $df=1$ )= $0.88$ ,  $p=0.35$ ; CFI=1.000, TLI=1.002, RMSEA=0.000

\*  $p<0.05$ . \*\*  $p<0.01$ . \*\*\*  $p<0.001$

**Table 3** Predictors of latent intercept and linear slope growth factors of child externalizing and internalizing behaviors across ages 18 to 54 months ( $n=346$ )

Predictors	EXT-intercept		EXT-slope		INT-intercept		INT-slope	
	B (SE)	$\beta$ (95 % CI)	B (SE)	$\beta$ (95 % CI)	B (SE)	$\beta$ (95 % CI)	B (SE)	$\beta$ (95 % CI)
AM depressive	0.20 (0.09)	0.15 (0.03–0.28)*	0.04 (0.04)	0.09 (–0.10–0.28)	0.10 (0.05)	0.13 (0.002–0.27)*	0.02 (0.03)	0.07 (–0.15–0.29)
AM antisocial	0.39 (0.13)	0.19 (0.07–0.31)**	–0.09 (0.06)	–0.15 (–0.34–0.05)	0.26 (0.08)	0.23 (0.10–0.35)***	–0.02 (0.05)	–0.05 (–0.15–0.29)
BM MDD	1.73 (0.72)	0.16 (0.03–0.29)*	0.07 (0.34)	0.02 (–0.17–0.22)	0.55 (0.42)	0.09 (–0.05–0.22)	0.26 (0.26)	0.11 (–0.12–0.35)
BM antisocial	1.09 (0.66)	0.11 (–0.02–0.24)	–0.29 (0.29)	–0.09 (–0.28–0.09)	0.83 (0.39)	0.15 (0.01–0.28)*	–0.34 (0.23)	–0.16 (–0.39–0.07)

EXT-model:  $\chi^2$  ( $n=346$ ;  $df=5$ )=4.66,  $p=0.46$ ; RMSEA=0.000; CFI=1.000; TLI=1.004; correlation between EXT-intercept and EXT-slope=–0.23,  $p<0.05$

INT-model:  $\chi^2$  ( $n=346$ ;  $df=5$ )=7.46,  $p=0.19$ ; RMSEA=0.038; CFI=0.988; TLI=0.965; correlation between INT-intercept and INT-slope=–0.04,  $p=0.83$

Correlations among predictors were equivalent for EXT and INT models: BM MDD with BM antisocial behavior=0.17,  $p<0.01$ , AM depressive symptoms with AM antisocial behavior=0.29,  $p<0.001$ , BM MDD with AM depressive symptoms=0.004, BM MDD with AM antisocial behavior=0.04, BM antisocial behavior with AM depressive symptoms=–0.04, BM antisocial behavior with AM antisocial behavior=–0.04.

EXT Child externalizing behaviors, INT Child internalizing behaviors, AM Adoptive mother, BM Biological mother, MDD major depressive disorder history, SE standard error. 95 % CI 95 % confidence interval

\* $p<0.10$ . \*\*  $p<0.05$ . \*\*\*  $p<0.01$ . \*\*\*\*  $p<0.001$

Child gender differences in patterns of association were not hypothesized. Still, we compared models with the associations between predictors and the problems constrained to be equal between genders and then relaxed; a model  $\Delta\chi^2/\Delta df > 3.84$  was used to determine that a model fit was improved or worsened. For externalizing intercepts, the fits of the constrained [ $\chi^2$  ( $df=18$ )=14.87] and unconstrained [ $\chi^2$  ( $df=10$ )=11.54] models did not differ significantly ( $\Delta\chi^2/\Delta df=0.42$ ), nor did the constrained [ $\chi^2$  ( $df=18$ )=27.77] and unconstrained [ $\chi^2$  ( $df=10$ )=10.05] models for internalizing intercepts ( $\Delta\chi^2/\Delta df=2.21$ ).

*Replication Using Adoptive Fathers' Reports* Adoptive mothers' and fathers' reports were correlated across problem type and assessment wave and ranged from  $r=0.29$  to  $0.49$ ,  $p<0.001$ . However, adoptive fathers reported lower levels of externalizing and internalizing behaviors across all waves than adoptive mothers did when rating the same child (paired  $t$ -tests ranged from  $t=1.86$ ,  $p=0.06$  to  $t=4.08$ ,  $p<0.001$ ). As shown in Table 2, unconditional growth models using adoptive fathers' reports were similar to those using adoptive mothers' reports. Multivariate models parallel to those in Table 3 were run using adoptive fathers' reports on the child outcomes. Genetic pathways were not replicated. However, adoptive mothers' depressive symptoms remained associated with externalizing behavior intercepts,  $\beta=0.20$ ,  $p<0.01$ . Paths from adoptive mothers' depressive symptoms and antisocial behaviors to internalizing behavior intercepts were significant in univariate,  $\beta=0.15$ , and  $0.21$ , respectively,  $p<0.05$ , but were only marginally significant in multivariate models,  $\beta=0.14$  and  $0.13$ , respectively,  $p=0.06$ . All subsequent models utilized adoptive mothers' reports on child externalizing and internalizing outcomes.

**Step 3: Probing Genetic Pathways for Mediation and Moderation**

Next, we tested whether non-genetic transmission mechanisms might better explain apparent genetic paths identified in Step 2. Specifically, we first questioned whether effects of biological mothers' MDD and antisocial behavior on child outcomes might be explained by the greater tendency for children of these mothers to have been exposed to detrimental prenatal and perinatal conditions (mediation). Second, we considered whether effects of biological mothers' symptoms and behaviors on child outcomes might depend on (be moderated by) the degree of adoption openness.

The first of these models regressed child outcome intercepts and slopes (run separately for child

externalizing and internalizing behaviors) on biological mothers' MDD and antisocial behavior and controlled for adoptive mothers' depressive symptoms and antisocial behavior. Biological mothers' MDD and antisocial behavior were independently associated with greater obstetric complications,  $\beta=0.17$  and  $0.15$ , respectively,  $p<0.01$ . However, there was no evidence of mediation. Specifically, the total significant effect of biological mothers' MDD on externalizing behavior intercept in this model,  $\beta=0.14$ ,  $p<0.05$ , was wholly direct,  $\beta=0.14$ ,  $p<0.05$ , and undiminished by the nonsignificant indirect effect via complications,  $\beta=-0.002$ ,  $p=0.88$ . Likewise, the total significant effect of biological mothers' antisocial behaviors on internalizing behavior intercept,  $\beta=0.14$ ,  $p<0.05$ , could be decomposed into a marginally significant (though essentially unchanged) direct effect,  $\beta=0.13$ ,  $p=0.06$ , and a nonsignificant indirect effect via obstetric complications,  $\beta=0.007$ ,  $p=0.47$ .

Using the same model controls as above, we next examined whether the association between biological mothers' lifetime histories of MDD and children's externalizing behavior intercept was explained by mothers' depressive symptoms during the prenatal period. As our modeling approach did not permit mediation by a binary variable, we simply controlled for prenatal symptoms. Biological mothers' prenatal symptoms covaried with their lifetime MDD histories ( $r=0.30$ ,  $p<0.001$ ), which was not surprising given the temporal and content overlap of these variables. The effect of biological mothers' MDD on externalizing behavior intercept was modestly reduced (from  $\beta=0.16$ ), and remained marginally significant,  $\beta=0.12$ ,  $p=0.07$ , whereas the prenatal depressive symptoms was significantly associated with the externalizing intercept,  $\beta=0.13$ ,  $p<0.05$ . Thus, findings indicated that biological mothers' lifetime MDD histories and prenatal depressive symptoms were associated with higher child externalizing behaviors, but did not suggest that this latter path substantially accounted for the former one.

Parallel analyses were conducted for the internalizing behavior outcomes. Biological mothers' prenatal depressive symptoms were modestly associated with their antisocial behaviors ( $\beta=0.10$ ,  $p=0.07$ ), but were not associated with child internalizing intercept ( $\beta=0.01$ ,  $p>0.10$ ), and did not diminish the magnitude or significance of the associations that any of the previously significant predictors had with this outcome.

With respect to moderation of genetic paths (from models in Table 3) by adoption openness,

we reran the growth model for externalizing behaviors, and regressed intercepts on biological mothers' MDD history, adoption openness, and the interaction; the latter term was not significant,  $\beta=0.07$ ,  $p=0.28$ . We then conducted a similar model for effects of biological mothers' antisocial behaviors on child internalizing behaviors. Internalizing behavior intercept was not associated with the interaction,  $\beta=-0.04$ ,  $p=0.64$ . Thus, there was no evidence that associations between biological mothers' symptoms/behaviors and child outcomes were stronger when there was greater post-adoption openness and contact.

#### Exploratory Analyses: Specificity of Prediction to Externalizing and Internalizing Behaviors

Next, we conducted a parallel process growth model for both child externalizing and internalizing behaviors. The unconditional model adequately fit the data; Model  $\chi^2$  ( $n=346$ ,  $df=6$ ) = 1.16,  $p=0.98$ ; CFI = 1.000, TLI = 1.017, RMSEA = 0.000. Intercepts were correlated ( $r=0.68$ ,  $p<0.001$ ), as were slopes ( $r=0.69$ ,  $p<0.01$ ); time-specific externalizing and internalizing behaviors residuals at 18, 27, and 54 months were allowed to correlate, and did so at  $rs=0.16$  ( $p=0.26$ ),  $0.50$ , and  $0.64$  ( $ps<0.01$ ), respectively.<sup>2</sup> Externalizing slope was predicted by internalizing intercept ( $\beta=-0.30$ ,  $p<0.01$ ), but internalizing slope was not associated with externalizing intercept ( $\beta=-0.01$ ,  $p=0.93$ ). As an initial check on robustness of the model, the four maternal psychopathology measures were added to this model as correlated predictors. Relative to findings reported in Table 3, predictive paths showed identical patterns of significance and were highly similar in magnitude; specifically, all paths to outcome intercepts were within  $\beta\pm 0.01$ , and no significant predictive paths to outcome slopes were found.

Next, the model was modified by regressing the externalizing behavior intercept on the internalizing intercept (i.e., making the correlation directional), and the four maternal psychopathology measures. Then, the same model was run for internalizing behavior intercept but controlling for (regressed on) the externalizing behavior intercept. In the interests of maximizing parsimony and statistical power, we did not model the mediators and moderators considered previously, and, as no observed predictors were associated with slope in prior analyses, we did not model these predictive paths.

<sup>2</sup> Observed scores correlated  $r=0.51$ ,  $p<0.001$  at 18 months; the relatively low correlation between the residuals likely is due to the fact that the latent intercepts (which were strongly correlated) were centered at 18 months.



*Specificity of Risk for Externalizing Behaviors* With a directional path from internalizing intercept controlled,  $\beta=0.63$ ,  $p<0.001$ , the paths from adoptive mothers' depressive symptoms and antisocial behavior to externalizing intercept were reduced considerably,  $\beta=0.08$  and  $0.04$ , respectively,  $p>0.10$ , from those reported in Table 3, and the previously significant path ( $\beta=0.16$ ,  $p<0.05$  in the Step 2 model) from biological mothers' MDD was diminished to  $\beta=0.10$ ,  $p=0.07$ . The previously nonsignificant path from biological mothers' antisocial behavior remained nonsignificant,  $\beta=0.03$ .

*Specificity of Risk for Internalizing Behaviors* Finally, the model was rerun with the directional path from the intercept of externalizing behaviors to internalizing behaviors controlled,  $\beta=0.63$ ,  $p<0.001$ . Adoptive mothers' antisocial behaviors persisted as a significant predictor of internalizing behavior intercept,  $\beta=0.12$ ,  $p<0.05$ , though it was reduced in magnitude relative to the path in Table 3 ( $\beta=0.23$ ,  $p<0.001$ ); paths from adoptive mothers' depressive symptoms ( $\beta=0.03$ ), and biological mothers' antisocial behaviors ( $\beta=0.05$ ) and MDD ( $\beta=0.002$ ; not previously significant) were reduced in magnitude and significance ( $p>0.10$ ) relative to those reported in Table 3.

In sum, risk associated with biological mothers' MDD was partially specific to child externalizing behaviors ( $p<0.10$ ), and risk associated with adoptive mothers' antisocial behavior was partially specific to child internalizing behaviors ( $p<0.05$ ). None of the other significant paths reported in Table 3 were found to reflect child outcome-specific risk.

## Discussion

Using an adoption design, we found that in general, adoptive mothers' depressive symptoms and antisocial behavior were independently associated with child behavioral dimensions at age 18 months. As adoptive mothers and their children were not biologically related, these associations may reflect environmental pathways by which mothers' depressive symptoms and antisocial behavior impact these child behaviors. This interpretation, rather than one based primarily on shared method variance and informant bias (Müller et al. 2011), is supported by several findings. Foremost, several of these pathways were replicated when adoptive mothers' symptoms and behaviors were considered in relation to adoptive fathers' reports of the child outcomes. Second, one would expect the modeling of one domain of adoptive mothers' psychopathology (e.g., depressive symptoms) to largely control for and diminish any association the other domain of adoptive mothers' psychopathology had with child outcomes if the associations were purely artifactual. However, in general the two dimensions independently predicted child outcomes and these paths were not markedly different in univariate versus

multivariate models. Third, if associations between adoptive mothers' symptoms and those of their children exclusively reflected shared method variance, then partialling out this association should have improved the strength of genetic path coefficients in multivariate models, which it did not. Still, this potential method artifact cannot be ruled out, and these findings require replication.

Biological mothers' histories of MDD and adult antisocial behavior also were found to confer risk for child externalizing and internalizing behaviors at age 18 months. These associations persisted when obstetric complications and depressive symptoms during the prenatal period were controlled. Prior studies indicate that adverse prenatal and perinatal conditions and events are associated with child behavioral and emotional maladjustment. In the present study, biological mothers' psychopathology was indeed associated with obstetric complications, and child prenatal exposure to maternal depressive symptoms was linked with externalizing behaviors at age 18 months. However, there was no evidence that these explanations accounted for the direct pathways of influence from biological mothers' psychopathology to the child outcomes. Interestingly, several studies of older children have found that perinatal complications were only associated with problem behavior for children experiencing other contextual adversity (Arseneault et al. 2002; Beck and Shaw 2004). Adoption may prevent some children from experiencing the number or combination of biopsychosocial risks necessary to negatively impact behavioral adaptation.

We also further probed the associations between biological mothers' psychopathology and child outcomes to determine whether they depended on the extent of contact and openness between adoptive and biological families. Such a result might imply that the association was environmentally rather than genetically mediated. However, there was no such evidence. It is worth highlighting that because biological mothers did not contribute reports of child outcomes, these associations are wholly immune to concerns of shared method variance and informant bias that plague many studies in developmental psychopathology. Primary findings as well as the null findings regarding mediation and moderation require replication, but are most consistent with the interpretation that the reported associations reflect genetic pathways of parent–child transmission by early childhood.

Taken together, the above findings support the broad conclusion that depressive and antisocial dimensions of maternal psychopathology confer independent risk for the early child behavioral outcomes considered here. This is consistent with recent factor analytic, epidemiologic, and genetic studies that all point to two distinct, heritable liabilities underlying most comorbidity among psychiatric syndromes: an internalizing and externalizing factor (Kendler et al. 2003; Kessler et al. 2011; Krueger and Markon 2006).

In contrast to our detection of pathways that were unique to *predictors* in our models, we found only modest evidence for specificity of pathways to the *outcomes*. Specifically, biological mothers' MDD history conferred some risk that was specific to child externalizing behaviors (though the path remained only marginally significant), and, furthermore, some risk associated with adoptive mothers' antisocial behaviors was specific to child internalizing behaviors. We emphasize that these outcome-specific paths were modest, and that overall our models supported general rather than problem-specific risk for child externalizing and internalizing behaviors. These findings do not necessarily contradict those of the studies reviewed above on distinct externalizing and internalizing liabilities. Rather, distinctions in the transmission mechanisms implicated in externalizing versus internalizing behaviors may not have been apparent in the toddler period perhaps because child problems are not yet fully differentiated or differentiable (Gilliom and Shaw 2004; Gjone and Stevenson 1997; van den Oord et al. 2000). With respect to the generalized rather than specific effects of adoptive mothers' psychopathology, we note that maternal depressive symptoms and antisocial behavior may be associated with different aspects of suboptimal caregiving environments, but those environments may be similarly associated with the two child outcomes. Given the modest strength of these paths, statistical power may have limited our ability to discern child problem-specific effects. Our examination of only two forms of maternal psychopathology also may have precluded our detection of distinct genetic vulnerabilities. We continue to emphasize the importance of discovering when and how these distinctive liabilities develop and become evident behaviorally. In all, our findings suggest this may occur later than age 18 months.

Contrary to our hypotheses, maternal psychopathology reported to age 18 months was not associated with subsequent changes in children's behaviors to 54 months of age. This may be partially explained by the modest variability observed in outcome slopes, and by the unexpected increases observed in externalizing behaviors over time (e.g., Gilliom and Shaw 2004). These increases may relate to sampling and exclusion criteria that yielded a sample with initially relatively low means that subsequently increased toward the population mean. Effects on slopes may become more detectable over time, when some children's behaviors begin to deviate more significantly from normative trajectories.

Of note, adoptees also may show different problem behavior trajectories than nonadopted samples due to differences in biological liabilities (genetic and prenatal exposure risk) and experiences related to their emerging understanding of their adoption (e.g., Juffer 2006). In Juffer and van IJzendoorn's (2005) meta-analysis of studies of more than 100,000 adopted and nonadopted individuals, adoptees showed higher levels of externalizing and internalizing problems than nonadopted controls, and domestic adoptees showed higher levels of both

problems than international adoptees. The findings are not wholly applicable to the present study participants as only 7 % of studies considered by Juffer and van IJzendoorn assessed youth prior to age 5 years, and child age at adoptive placements was greater than 12 months in more than half of the studies. Still, this may help explain why participants in the present study showed different behavior trajectories than children in other developmental studies. The increases in externalizing behaviors noted in the present study may inform studies of the development of adopted children. Such studies often cannot discern the effects of adoption on child development from the confounded effects of prenatal complications and early adversity. Although there was no comparison group of nonadopted children, the present study design and inclusion/exclusion criteria suggest the increases in externalizing behaviors observed here are not attributable to those risks.

### Study Limitations

Several study limitations should be noted. First, low biological fathers' participation precluded consideration of their influences in these models. Thus, genetic effects were underestimated and could not be conclusively distinguished from potential unmeasured impacts of biological mothers' psychopathology on the intrauterine environment. It also was not possible to compare the effects of paternal and maternal psychopathology on risk for problems in offspring, as has been done in some prior studies that did not use genetically informed designs (Kim et al. 2009; Koop and Beauchaine 2007). Second, genetic pathways were not replicated using adoptive fathers' reports of child outcomes. Given that fathers' reports were systematically lower than mothers', it is possible that restricted variance decreased statistical power to detect these effects. Third, psychiatric diagnoses of adoptive parents were not available. Fourth, relatively high levels of functioning and contextual stability are required for parents to adopt children, a point that was evident in the relatively low prevalence of clinically significant depressive symptoms and antisocial behaviors among adoptive mothers. Thus, although adoptive parent characteristics predict child outcomes in this and other studies using this sample (Pemberton et al. 2010), restricted range may limit generalizability of environmental pathways to families in more deprived environments. Fifth, given differences in base rates and measures of depression and antisocial behavior in biological versus adoptive mothers, the strengths of genetic and environmental paths cannot be compared. Sixth, we cannot be certain of the direction of the influence between child behaviors and adoptive parent characteristics when children were age 18 months. Sixth, child exclusion criteria limit the generalizability of estimates of the influences of obstetric complications on child outcomes. Finally, the present study supports additive genetic and environmental influences; examining interacting influences of maternal

psychopathology or even more dynamic interplay over development is essential but was beyond the scope of this study. Similarly, examination of potential mediators of the association between parent and child psychopathology (e.g., parenting) was not considered here but is an important next step for future research in this area.

### Conclusions and Implications

Findings support the existence of independent genetic and environmental influences of parent depressive symptoms and antisocial behavior on child behavioral outcomes. Such pathways are widely assumed to exist but have rarely been examined using requisite genetically-informed designs, especially with respect to the very early manifestations of risk transmission. Present findings indicate problem transmission pathways are detectable by age 18 months but may not yet be specific to child externalizing versus internalizing behaviors. Direct genetic effects suggest the value of preventive intervention for children born to mothers with histories of psychiatric problems. Direct environmental effects suggest genetic risks may be partially offset by the caregiving environments provided by psychologically healthy parents, and uphold that interventions directed at parent psychopathology is a promising avenue for improving child behavioral trajectories (e.g., Garber et al. 2011).

**Acknowledgements** This study was supported by grant R01 HD042608 from NICHD, NIDA, and OBSSR, NIH, U.S. PHS (PIs: Reiss and Leve), by grant R01 DA020585 from NIDA, NIMH, and OBSSR, NIH, U.S. PHS (PI: Neiderhiser), by grant R01 MH092118 from NIMH, NIH, U.S. PHS (PIs: Neiderhiser and Leve). 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 are very grateful to the adoption agencies and families who participated in the study. We thank Xiaojia Ge, John Reid, Rand Conger, Laura Scaramella, and Jody Ganiban for their scientific contributions to the larger study and Michelle Baumann for her editorial assistance.

### References

- Achenbach, T. M., & Rescorla, L. A. (2000). *Manual for the ASEBA preschool forms & profiles*. Burlington: University of Vermont, Research Center for Children, Youth, & Families.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington: Author.
- Angold, A., & Costello, E. J. (1993). Depressive comorbidity in children and adolescents: empirical, theoretical, and methodological issues. *The American Journal of Psychiatry*, *150*, 1779–1791.
- Arseneault, L., Tremblay, R. E., Boulerice, B., & Saucier, J. F. (2002). Obstetrical complications and violent delinquency: testing two developmental pathways. *Child Development*, *73*, 496–508.
- Beardslee, W. R., Versage, E. M., & Gladstone, T. R. G. (1998). Children of affectively ill parents: a review of the past 10 years. *Journal of the American Academy of Child and Adolescent Psychiatry*, *37*, 1134–1141.
- Beck, A., & Shaw, D. S. (2004). The influence of perinatal complications and environmental adversity on boys' antisocial behavior. *Journal of Child Psychology and Psychiatry*, *46*, 35–46.
- Beck, A., & Steer, R. (1993). *Beck depression inventory manual*. The Psychological Corporation.
- Beck, A. T., Steer, R. A., & Garbin, M. G. J. (1988). Psychometric properties of the Beck depression inventory twenty-five years of evaluation. *Clinical Psychology Review*, *8*, 77–100.
- Blouin, A. G., Perez, E. L., & Blouin, J. H. (1988). Computerized administration of the diagnostic interview schedule. *Psychiatry Research*, *23*, 335–344.
- Brennan, P. A., Pargas, R., Walker, E. F., Green, P., Newport, D. J., & Stowe, Z. (2008). Maternal depression and infant cortisol: influences of timing, comorbidity and treatment. *Journal of Child Psychology and Psychiatry*, *49*, 1099–1107.
- Conger, R. D., Belsky, J., & Capaldi, D. M. (2009). The intergenerational transmission of parenting: closing comments for the special section. *Developmental Psychology*, *45*, 1276–1283.
- Cottler, L. B., Compton, W. M., Ridenour, T. A., Abdallah, A. B., & Gallagher, T. (1998). Reliability of self-reported antisocial personality disorder symptoms among substance abusers. *Drug and Alcohol Dependence*, *49*, 189–199.
- Elkins, I. J., Iacono, W. G., Doyle, A. E., & McGue, M. (1997). Characteristics associated with the persistence of antisocial behavior: results from recent longitudinal research. *Aggression and Violent Behavior*, *2*, 101–124.
- Garber, J., Ciesla, J. A., McCauley, E., Diamond, G., & Schloretd, K. A. (2011). Remission of depression in parents: links to healthy functioning in their children. *Child Development*, *82*, 226–243.
- Gilliom, M., & Shaw, D. S. (2004). Codevelopment of externalizing and internalizing problems in early childhood. *Developmental Psychopathology*, *16*, 313–333.
- Gjone, H., & Stevenson, J. (1997). The association between internalizing and externalizing behavior in childhood and early adolescence: genetic or environmental common influences? *Journal of Abnormal Child Psychology*, *25*, 277–286.
- Greenberg, P. E., Kessler, R. C., Bimbaum, H. G., Leong, S. A., Lowe, S. W., Berglund, P. A., & Corey-Liske, P. K. (2003). The economic burden of depression in the United States: how did it change between 1990 and 2000? *The Journal of Clinical Psychiatry*, *64*, 1465–1475.
- Harold, G. T., Rice, F., Hay, D. F., Boivin, J., van den Bree, M., & Thapar, A. (2011). Familial transmission of depression and antisocial behavior symptoms: disentangling the contribution of inherited and environmental factors and testing the mediating role of parenting. *Psychological Medicine*, *41*, 1175–1178.
- Hicks, B. M., Krueger, R. F., Iacono, W. G., McGue, M., & Patrick, C. J. (2004). Family transmission and heritability of externalizing disorders: a twin-family study. *Archives of General Psychiatry*, *61*, 922–928.
- Juffer, F. (2006). Children's awareness of adoption and their problem behavior in families with 7-year-old internationally adopted children. *Adoption Quarterly*, *9*, 1–22.
- Juffer, F., & van Ijzendoorn, M. H. (2005). Behavior problems and mental health referrals of international adoptees: a meta-analysis. *Journal of the American Medical Association*, *293*, 2501–2515.
- Kendler, K. S., Prescott, C. A., Myers, J., & Neale, M. C. (2003). The structure of genetic and environmental risk factors for common psychiatric and substance use disorders in men and women. *Archives of General Psychiatry*, *60*, 929–937.
- Kessler, R. C., & Üstün, T. B. (2004). The World Mental Health (WMH) survey initiative version of the World Health Organization (WHO) Composite International Diagnostic Interview (CIDI). *The*

- International Journal of Methods in Psychiatry Research*, 13, 93–121.
- Kessler, R.C., Ormel, J., Petukhova, M., McLaughlin, K.A., Green, J.G., Russo, L.J., ...Sagar, R., & Ustün, T.B. (2011). Development of lifetime comorbidity in the World Health Organization world mental health surveys. *Archives of General Psychiatry*, 68, 90–100.
- Kim, H. K., Capaldi, D. M., Pears, K. C., Kerr, D. C. R., & Owen, L. D. (2009). Intergenerational transmission of internalising and externalising behaviours across three generations: gender-specific. *Criminal Behavior and Mental Health*, 19, 125–141.
- Kim-Cohen, J., Moffitt, T. E., Taylor, A., Pawlby, S. J., & Caspi, A. (2005). Maternal depression and children's antisocial behavior. *Archives of General Psychiatry*, 62, 173–181.
- Koop, L. M., & Beauchaine, T. P. (2007). Patterns of psychopathology in the families of children with conduct problems, depression, and both psychiatric conditions. *Journal of Abnormal Child Psychology*, 35, 301–312.
- Krueger, R. F., & Markon, K. E. (2006). Reinterpreting comorbidity: a model-based approach to understanding and classifying psychopathology. *Annual Review of Clinical Psychology*, 2, 111–133.
- Leve, L.D., Neiderhiser, J.M., Shaw, D.S., Ganiban, J., Natsuaki, M.N., & Reiss, D. (2013). The Early Growth and Development Study: A prospective adoption study of child behavior from birth through middle childhood. *Twin Research and Human Genetics*.
- Levenson, M. R., Kiehl, K. A., & Fitzpatrick, C. M. (1995). Assessing psychopathic attributes in a non-institutionalized population. *Journal of Personality and Social Psychology*, 68, 151–158.
- Lewis, G., Rice, F., Harold, G. T., Collishaw, S., & Thapar, A. (2011). Investigating environmental links between parent depression and child depressive/anxiety symptoms using an assisted conception design. *Journal of the American Academy of Child and Adolescent Psychiatry*, 50, 451–459.
- Mathiesen, K. S., & Sanson, A. (2000). Dimensions of early childhood behavior problems: stability and predictors of change from 18 to 39 months. *Journal of Abnormal Child Psychology*, 28, 15–31.
- McNeil, T., & Sjöström, K. (1995). *McNeil-Sjöström scale for obstetric complications*. Sweden: Lund University.
- Meyer, J.M., Rutter, M., Silberg, J.L., Maes, H.H., Simonoff, E., Shillady, L.L., ... Eaves, L.J. (2000). Familial aggregation for conduct disorder symptomatology: The role of genes, marital discord and family adaptability. *Psychological Medicine*, 30, 759–774.
- Müller, J. M., Achtergarde, S., & Furniss, T. (2011). The influence of maternal psychopathology on ratings of child psychiatric symptoms: an SEM analysis on cross-informant agreement. *European Child & Adolescent Psychiatry*, 20, 241–252.
- Murray, L., Arteche, A., Fearon, P., Halligan, S., Goodyer, I., & Cooper, P. (2011). Maternal postnatal depression and the development of depression in offspring up to 16 years of age. *Journal of the American Academy of Child and Adolescent Psychiatry*, 50, 460–470.
- Muthén, L., & Muthén, B. (1998–2010). *Mplus user's guide*. Los Angeles, CA: Author.
- O'Connor, T. G., McGuire, S., Reiss, D., Hetherington, E. M., & Plomin, R. (1998). Co-occurrence of depressive symptoms and antisocial behavior in adolescence: a common genetic liability. *Journal of Abnormal Psychology*, 107, 27–37.
- Olino, T. M., Pettit, J. W., Klein, D. N., Allen, N. B., Seeley, J. R., & Lewinsohn, P. M. (2008). Influence of parental and grandparental major depressive disorder on behavior problems in early childhood. A three-generational study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47, 53–60.
- Pemberton, C. K., Neiderhiser, J. M., Leve, L. D., Natsuaki, M. N., Shaw, D. S., Reiss, D., & Ge, X. (2010). Influence of parental depressive symptoms on adopted toddler behaviors: an emerging developmental cascade of genetic and environmental effects. *Development and Psychopathology*, 22, 803–818.
- Scaramella, L. V., & Leve, L. D. (2004). Clarifying parent-child reciprocities during early childhood: the early childhood coercion model. *Clinical Child and Family Psychology Review*, 7, 89–107.
- Shaw, D. S., Gilliom, M., & Giovannelli, J. (2000). Aggressive behavior disorders. In C. H. Zeanah (Ed.), *Handbook of infant mental health* (2nd ed., pp. 397–411). New York: Guilford.
- Silberg, J. L., Maes, H., & Eaves, L. J. (2010). Genetic and environmental influences on the transmission of risk from parental depression to children's depression and conduct problems. *Journal of Child Psychology and Psychiatry*, 51, 734–744.
- Sullivan, P. F., Neale, M. C., & Kendler, K. S. (2000). Genetic epidemiology of major depression: review and meta-analysis. *The American Journal of Psychiatry*, 157, 1552–1562.
- Tully, E. C., Iacono, W. G., & McGue, M. (2008). An adoption study of parental depression as an environmental liability for adolescent depression and childhood disruptive disorders. *The American Journal of Psychiatry*, 165, 1148–1154.
- van den Oord, E. J. C. G., Boomsma, D. I., & Verhulst, F. C. (2000). A study of genetic and environmental effects on the co-occurrence of problem behaviors in three-year-old-twins. *Journal of Abnormal Psychology*, 109, 360–372.
- van der Valk, J. C., van den Oord, E. J. C. G., Verhulst, F. C., & Boomsma, D. I. (2003). Genetic and environmental contributions to stability and change in children's internalizing and externalizing problems. *Journal of the American Academy of Child and Adolescent Psychiatry*, 42, 1212–1220.
- Welsh, B. C., Loeber, R., Stevens, B. R., Stouthamer-Loeber, M., Cohen, M. A., & Farrington, D. P. (2008). The costs of juvenile crime in urban areas: a longitudinal perspective. *Youth Violence and Juvenile Justice*, 6, 3–27.