Influences of Biological and Adoptive Mothers' Depression and Antisocial Behavior on

Adoptees' Early Behavior Trajectories

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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.

Keywords: depression, externalizing, internalizing, early childhood, genetic risk, adoption.

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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 & Costello, 1993;

Kendler, Prescott, Myers, & Neale, 2003; Krueger & Markon, 2006). Although distinct

externalizing (e.g., aggression and oppositionality) and internalizing (e.g., anxiety, sadness)

factors are difficult to discern during toddlerhood (Gjone & Stevenson, 1997; van den Oord,

Boomsma, & Verhulst, 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, Belsky, & Capaldi, 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, Versage, & Gladstone, 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, Iacono, & McGue, 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, Rice, Harold, Collishaw, & Thapar, 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, Maes, & Eaves, 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, Krueger, Iacono, McGue, & Patrick, 2004; Sullivan, Neale, & Kendler, 2000; van der Valk, van den Oord, Verhulst, & Boomsma, 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, McGuire,

Reiss, Hetherington, & Plomin, 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,

Moffitt, Taylor, Pawlby, & Caspi, 2005). Others have found maternal internalizing symptoms to

be associated with both child externalizing and internalizing symptoms (Kim, Capaldi, Pears,

Kerr, & Owen, 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 & 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 & 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 & 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 & 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., in press). Children from a range of racial backgrounds (58.4% White) participated. Adoptive parents[1] were predominantly Caucasian (92%), educated (mean level = "completed college"), middle-class

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[1] 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."

(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).

Measures

Adoptive mothers' depressive symptoms. Mothers completed the self-reported Beck Depression Inventory (BDI; Beck & Steer, 1993) when children were age 18 months ($\alpha = .80$). 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 administrated in the present study. Thus, mothers completed 20 items from this 21-item scale, and total scores could range from 0–60. 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, Steer, & Garbin, 1988).

Adoptive mothers' antisocial behavior. Mothers completed an adaptation of the Antisocial Action Scale (Levenson, Kiehl, & Fitzpatrick, 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 ($\alpha = .56$) using a 4-point Likert scale ranging from 1 (*never*) to 4 (*often*); therefore, total scores could range from 13–52.

Biological mothers' lifetime history of MDD. At 18 months postpartum, mothers completed the Composite International Diagnostic Interview (Kessler & 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 $\kappa = .45$ to .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, Perez, & Blouin, 1988) at 18 months postpartum. Others have found test-retest reliability for the ASPD diagnosis acceptable (e.g., $\kappa = .527$, Cottler, Compton, Ridenour, Abdallah, & Gallagher, 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, Iacono, Doyle, & McGue, 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 0–3); 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 & Sjöström, 1995).

Child externalizing and internalizing behavior. Adoptive mothers and fathers independently completed the Child Behavior Checklist for ages 1½-5 years (Achenbach & 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 & 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, Gilliom, & Giovannelli, 2000). Raw scores were constructed identically across time (α = .71–.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 .73–.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 & 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 .05 was used as

the criterion for judging statistical significance; model fit was judged based on a χ^2 with a *p*-

value greater than .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.

<div align="center">Results</div>

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 note that externalizing behaviors at age 18 months were correlated with those at 27 and 54 months (r = .65 and .45, respectively, $p < .001$), as were internalizing behaviors ($r = .58$ and .39, respectively, $p < .001$), 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, $\beta = .18$; adoptive mothers' depressive symptoms, $\beta = .21$, and antisocial

behavior, $\beta = .23$, all $p < .05$; and marginally by biological mothers' antisocial behavior, $\beta = .12$,

$p = .08$. Internalizing behavior intercepts were predicted by biological mothers' antisocial

behavior, $\beta = .15$; adoptive mothers' depressive symptoms, $\beta = .20$; adoptive mothers' antisocial

behavior, $\beta = .30$, all $p < .05$; and marginally by biological mothers' MDD, $\beta = .12$, $p = .08$.

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

$\beta = .20$ to $\beta = .13$, ps < .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 ($r = .17$) and adoptive mothers ($r = .29$) and

yielded no concerns regarding multicollinearity. In the separate models by outcome, the four

predictors accounted for significant variance in externalizing and internalizing intercepts, $R^2 =$ .12 and .12, respectively, $p < .01$, but not in slopes, $R^2 = .03$ and .04, respectively.

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 [χ^2 ($df = 18$) = 14.87] and unconstrained [χ^2 ($df = 10$) = 11.54] models did not differ significantly ($\Delta\chi^2/\Delta df = .42$), nor did the constrained [χ^2 ($df = 18$) = 27.77] and unconstrained [χ^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 $r = .29$ to .49, $p < .001$. 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 $t = 1.86$, $p = .06$ to $t = 4.08$, $p < .001$). 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, $\beta = .20$, $p < .01$. Paths from adoptive mothers' depressive symptoms and antisocial behaviors to internalizing behavior intercepts were significant in univariate, $\beta = .15$, and .21, respectively, $p < .05$, but were only marginally significant in multivariate models, $\beta = .14$ and .13, respectively, $p = .06$. 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, $\beta = .17$ and $.15$, respectively, $p < .01$. However, there was no evidence of mediation. Specifically, the total significant effect of biological mothers' MDD on externalizing behavior intercept in this model, $\beta = .14$, $p < .05$, was wholly direct, $\beta = .14$, $p < .05$, and undiminished by the non-significant indirect effect via complications, $\beta = -.002$, $p = .88$. Likewise, the total significant effect of biological mothers' antisocial behaviors on internalizing behavior intercept, $\beta = .14$, $p < .05$, could be decomposed into a marginally significant (though essentially unchanged) direct effect, $\beta = .13$, $p = .06$, and a non-significant indirect effect via obstetric complications, $\beta = .007$, $p = .47$.

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 ($r = .30$, $p < .001$), 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 $\beta = .16$), and remained marginally significant, $\beta = .12$, $p = .07$, whereas

the prenatal depressive symptoms was significantly associated with the externalizing intercept, β

$= .13$, $p < .05$. 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

($\beta = .10$, $p = .07$), but were not associated with child internalizing intercept ($\beta = .01$, $p > .10$), 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, $\beta = .07$, $p = .28$. 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, $\beta = -.04$, $p = .64$. 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 χ^2 ($n = 346$, $df = 6$) = 1.16, p = .98; CFI = 1.000, TLI = 1.017, RMSEA = .000. Intercepts were correlated ($r = .68$, $p < .001$), as were slopes ($r = .69$, $p < .01$); time-specific externalizing and internalizing behaviors residuals at 18, 27, and 54 months were allowed to correlate, and did so at rs = .16 ($p = .26$), .50, and .64 (ps $< .01$), respectively.[2] Externalizing slope was predicted by internalizing intercept ($\beta = -.30$, $p < .01$), but internalizing slope was not associated with externalizing intercept ($\beta = -.01$, $p = .93$). 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 $\beta \pm .01$, 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.

Specificity of risk for externalizing behaviors. With a directional path from internalizing intercept controlled, $\beta = .63$, $p < .001$, the paths from adoptive mothers' depressive symptoms and antisocial behavior to externalizing intercept were reduced considerably, $\beta = .08$ and .04,

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[2] Observed scores correlated $r = .51$, $p < .001$ 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.

respectively, *p* > .10, from those reported in Table 3, and the previously significant path (β = .16, *p* < .05 in the Step 2 model) from biological mothers' MDD was diminished to β = .10, *p* = .07. The previously nonsignificant path from biological mothers' antisocial behavior remained nonsignificant, β = .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, β = .63, *p* < .001. Adoptive mothers' antisocial behaviors persisted as a significant predictor of internalizing behavior intercept, β = .12, *p* < .05, though it was reduced in magnitude relative to the path in Table 3 (β = .23, *p* < .001); paths from adoptive mothers' depressive symptoms (β = .03), and biological mothers' antisocial behaviors (β = .05) and MDD (β = .002; not previously significant) were reduced in magnitude and significance (*p* > .10) relative to those reported in Table 3.

In sum, risk associated with biological mothers' MDD was partially specific to child externalizing behaviors (*p* < .10), and risk associated with adoptive mothers' antisocial behavior was partially specific to child internalizing behaviors (*p* < .05). None of the other significant paths reported in Table 3 were found to reflect child outcome-specific risk.

<div align="center">Discussion</div>

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, Achtergarde, &

Furniss, 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, Tremblay, Boulerice, & Saucier, 2002; Beck & 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 that 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 & 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 & Shaw, 2004; Gjone & 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 & 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 non-adopted 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 non-adopted 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 non-adopted 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; Kopp & 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, Ciesla, McCauley, Diamond, & Schloredt, 2011).

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Table 1

Descriptive Statistics for Study Predictors and Controls

	Mean (SD) or % positive	Range
AM depressive symptoms (*n* = 333)	3.86 (3.86)	0–25
AM antisocial behavior (*n* = 337)	16.90 (2.47)	13–28
BM MDD lifetime history (*n* = 304)	29.6%	0–1
BM adult antisocial behavior (*n* = 295)	45.4%	0–1
BM prenatal depressive symptoms (*n* = 343)	62.1%	0–1
Obstetric complications (*n* = 346)	10.33 (6.98)	0–42
Adoption openness (*n* = 346)	.04 (.94)	-2.06–1.84

Note. AM = Adoptive mother, BM = Biological mother, MDD = major depressive disorder history.

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[a])				
EXT-18 months (0)	11.06 (0–31)	6.01	9.56 (0–30)	6.07
EXT-27 months (.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 (.31)***	25.27 (2.80)***	9.70 (.32)***	25.55 (2.98)***
EXT-slope	.58 (.14)***	2.36 (1.15)*	.48 (.13)***	2.49 (1.18)*
	Mother-reports		Father-reports	
Child INT Behaviors Model	*Mean (range)*	*SD*	*Mean (range)*	*SD*
Observed scores (growth weight[a])				

INT-18 months (0)	5.01 (0–24)	3.47	4.54 (0–23)	3.88
INT-27 months (.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 (.18)***	8.30 (1.06)***	4.59 (0–23)	10.63 (1.29)***
INT-slope	.90 (.11)***	1.31 (.61)*	.68 (.11)***	1.38 (.66)*

Note. [a]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 χ^2 ($n = 346$, $df = 1$) = .11, $p = .87$; CFI = 1.000, TLI = 1.011, RMSEA = .000. Father-report EXT Model χ^2 ($n = 334$, $df = 1$) = 1.37, $p = .24$; CFI = .998, TLI = .995, RMSEA = .033. Mother-report INT Model χ^2 ($n = 346$, $df = 1$) = .51, $p = .48$; CFI = 1.000, TLI = 1.008, RMSEA = .000. Father-report INT Model χ^2 ($n = 334$, $df = 1$) = .88, $p = .35$; CFI = 1.000, TLI = 1.002, RMSEA = .000.

* $p < .05$. ** $p < .01$. *** $p < .001$.

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)

	EXT-Intercept		EXT-slope		INT-Intercept		INT-slope	
Predictors	B (SE)	β (95% CI)	B (SE)	β (95% CI)	B (SE)	β (95% CI)	B (SE)	β (95% CI)
AM depressive	.20 (.09)	.15 (.03–.28) *	.04 (.04)	.09 (-.10–.28)	.10 (.05)	.13 (.002–.27)*	.02 (.03)	.07 (-.15–.29)
AM antisocial	.39 (.13)	.19 (.07–.31)**	-.09 (.06)	-.15 (-.34–.05)	.26 (.08)	.23 (.10–.35)***	-.02 (.05)	-.05 (-.15–.29)
BM MDD	1.73 (.72)	.16 (.03–.29)*	.07 (.34)	.02 (-.17–.22)	.55 (.42)	.09 (-.05–.22)	.26 (.26)	.11 (-.12–.35)
BM antisocial	1.09 (.66)	.11 (-.02–.24)	-.29 (.29)	-.09 (-.28–.09)	.83 (.39)	.15 (.01–.28)*	-.34 (.23)	-.16 (-.39–.07)

Note. 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.

EXT-model: χ^2 (n = 346; df = 5) = 4.66, p =.46; RMSEA = 0.000; CFI = 1.000; TLI = 1.004; correlation between EXT-intercept and EXT-slope = -.23, p < .05.

INT-model: χ^2 (n = 346; df = 5) = 7.46, p =.19; RMSEA = 0.038; CFI = .988; TLI = .965; correlation between INT-intercept and INT-slope = -.04, p = .83.

Correlations among predictors were equivalent for EXT and INT models: BM MDD with BM antisocial behavior = .17, p < .01, AM depressive symptoms with

AM antisocial behavior = .29, p < .001, BM MDD with AM depressive symptoms = .004, BM MDD with AM antisocial behavior = .04, BM antisocial behavior

with AM depressive symptoms = -.04, BM antisocial behavior with AM antisocial behavior = -.04.

†p < .10. * p < .05. ** p < .01. *** p < .001.