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Infant Pathways to Externalizing Behavior: Evidence of Genotype x Environment Interaction

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Abstract

To further the understanding of the effects of early experience on the pathways to externalizing behavior, 9-month-old infants were observed during a frustration task. The analytical sample was composed of 348 linked triads of participants (adoptive parents, adopted child, and birth parent[s]) from a prospective adoption study, thereby permitting an examination of the interplay between genetic characteristics and early environmental experiences. It was hypothesized that genetic risk for externalizing problems (indexed by birth parents' externalizing behavior) and affect dysregulation in the home (indexed by adoptive parents' anxious/depressive symptoms) would independently and interactively predict a known precursor to externalizing problems: heightened infant attention to frustrating events. The results from the SEM analyses supported the moderation hypotheses involving adoptive mother affect dysregulation but did not support those involving adoptive father affect dysregulation: infants at genetic risk showed heightened attention to frustrating events only when the adoptive mother had higher levels of anxious/depressive symptoms. The Genotype x Environment interaction pattern held when substance use during pregnancy was considered. Sex-specific patterns of effects and future directions are discussed.

The independent contributions of genetic and environmental influences to child behavior have been well-established over the past several decades using behavioral genetic designs (Reiss, Neiderhiser, Hetherington, & Plomin, 2000; Rutter, 2006). This body of research has indicated moderate genetic effects on most dimensions of externalizing behavior (Rhee & Waldman, 2002). Emanating from and building upon this work, there is a burgeoning body of evidence from behavioral and molecular genetic studies suggesting that environmental factors moderate or mediate genetic influences on problem behaviors. Studies of Genotype x Environment (GxE) interaction help to illustrate how inherited characteristics render some individuals more

susceptible to the negative or positive effects of specific environments (Moffitt, Caspi, & Rutter, 2005). This line of investigation is important in helping to identify mechanisms whereby specific environmental processes might offset or exacerbate genetic risk, thereby suggesting potential targets for preventive interventions. Most of the published studies in this area, however, have used retrospective measures of the environment or have studied environmental processes in middle childhood and beyond (Caspi et al., 2002; Feinberg, Button, Neiderhiser, Reiss, & Hetherington, 2007). Such approaches miss the opportunity to capture environmental experiences during the early childhood developmental period, which has been found to be malleable to preventive interventions (Olds, 2002; Shaw, Dishion, Supplee, Gardner, & Arnds, 2006). In this paper, we examine specific genetic and family environmental processes on a known precursor of externalizing behavior: infant heightened attention to frustrating events (Crockenberg, Leerkes, & Barrig Jo, 2008).

Connection Between Attention to Frustration and Subsequent Externalizing Behavior

The focus on infant attention to frustrating events, which has known links to subsequent externalizing behavior, was selected because infants are not developmentally capable of engaging in behaviors typically captured by standard definitions of externalizing behavior (e.g., cheating, lying, or directed aggression). Further, temperamental characteristics such as anger do not show a consistent relationship with externalizing problems until 12–24 months of age (Blair, 2002; Lyons-Ruth, Easterbrooks, & Cibelli, 1997; Smeeckens, Riksen-Walraven, & van Bakel, 2007). The behaviors involved in attention regulation undergo a developmental shift between 3 and 6 months of age (e.g., the acquisition of visual orientation skills and the ability to disengage visual gaze; Johnson, Posner, & Rothbart, 1991; Posner & Peterson, 1990), making infancy an ideal time to examine attentional processes. One aspect of attention—infant’s attention to frustrating events (defined in this paper as focus and persistence on a frustrating event)—has been examined in numerous studies during early childhood. Stifter and Braungart (1995) indicated that 5- and 10-month-olds were more likely to visually shift their attention away from a frustrating event when infant distress was decreasing, suggesting a regulatory role of infant ability to shift attention away from frustrating events. In addition, converging evidence from longitudinal studies has indicated a link between attentional focus on frustrating events and later externalizing problems.

In a longitudinal study of 5-month-old infants, Crockenberg et al. (2008) found that infants who failed to shift attention away from frustrating events (an arm restraint and a toy removal task), exhibited increased aggressive behavior at 2-½ years of age. Attention to frustration was defined by coder ratings of infant attempts to inspect the frustrating event and situation. The association between this infant behavior and later aggression was strongest when mothers encouraged their child to look at the frustrating event, which is consistent with the notion that maternal regulation processes moderate pathways to externalizing problems. Crockenberg and colleagues suggested that attention to a frustrating event is the dominant response and that successfully looking away reflects the ability to adaptively manage frustration. Regulation characteristics of the mother might undermine the development of a child’s capacity to shift attention away from frustrating events, inadvertently contributing to the development of externalizing problems. In the present study, we mirrored the Crockenberg et al. work by utilizing a similar toy removal task across repeated intervals and by coding for infant attention to the frustrating event and situation.

Although we are unaware of other studies that have employed toy removal protocols to measure attention to frustrating events, several longitudinal studies have examined early childhood attentional problems using other methods and have found similar associations with subsequent externalizing problems. For example, Caspi, Henry, McGee, Moffitt, and Silva (1995) found

that observer ratings of 3-year-olds' inability to modulate attention and lack of control were prospectively associated with externalizing problems in adolescence. Further, at 21 years of age, young children with these attention problems were more likely to be diagnosed with antisocial personality disorder (Caspi, Moffitt, Newman, & Silva, 1996). Tracing the pathways to externalizing problems earlier in development in two independent cohorts of low-income families, Shaw, Keenan, and Vondra (1994) and Shaw et al. (1998) observed mothers with their 12-month-olds during a task designed to elicit infant frustration behavior (i.e., high chair task). In both cohorts, boys who were higher on persistence in the face of caregiver unresponsivity at 12 months showed elevated rates of externalizing problems at age 2 and between ages 3 and 3-½ based on observations and maternal ratings of disruptive behavior (e.g., noncompliance, aggression). These studies replicated earlier work by Martin (1981), who used the same high chair task and found remarkably similar results for boys from less disadvantaged backgrounds.

There is also evidence to indicate that at-risk children may have heightened states of arousal and reactivity (Fisher, Stoolmiller, Gunnar, & Burraston, 2007), which might cause them to focus on negative cues that typically invoke negative emotions such as anger (Shackman, Shackman, & Pollak, 2007). Consistent with this body of work, we hypothesize that, under conditions of frustration, infants at risk for externalizing problems will react with increased attention and persistence. The extent to which the early home environment and genetic factors jointly affect the development of attention to frustration has yet to be investigated; in this study, we extended prior work by focusing on genetic and environmental influences on infants' attention behavior during a frustration task.

Genetic Contributions to Externalizing Behavior

The results from twin and adoption studies on the heritability of externalizing behavior provide evidence that the externalizing behaviors of interest in the proposed study are genetically influenced. In a meta-analysis of 24 genetically informative studies on aggression, Miles and Carey (1997) found that genetic effects accounted for approximately 50% of the variance in aggressive behavior. More recently, in a meta-analysis of 51 twin and adoption studies of antisocial behavior, Rhee and Waldman (2002) found more modest genetic influences (41%). The findings from recent twin and adoption studies also point to genetic influences on attention deficit problems (Thapar, O'Donovan, & Owen, 2005), novelty seeking (Hiroi & Agatsuma, 2005), and the associations between novelty seeking, externalizing behavior, and substance use (Laucht et al., 2007). Although useful in identifying potentially heritable aspects of externalizing behavior, such findings provide little information about the mechanisms of development, how the phenotype might evolve across development, and how genetic aspects of externalizing behavior during adolescence and adulthood relate to the genetic aspects of infants' propensity for externalizing behavior.

The findings from life-course studies by Laucht and colleagues have provided information about possible mechanisms in this regard. In this work, novelty seeking was found to be associated with a polymorphism of the DRD4 gene (Laucht, Becker, El-Faddagh, Hohm, & Schmidt, 2005). In a longitudinal study from infancy to adolescence, Laucht, Becker, and Schmidt (2006) found that visual exploratory behavior (i.e., longer initial looking times) and a higher response decrement (i.e., habituation) in 3-month-olds was associated with novelty seeking in adolescence and the 7r DRD4 allele variant. This is one of the few areas where a genetically influenced characteristic has been shown to have different and specific phenotypic expressions across development, manifested as attention during early infancy and as novelty seeking (a form of externalizing behavior) during adolescence. Guided by this genetically informed work and the phenotypic work on the association between attention to frustration and

later externalizing problems described above, we focused on infant attention to frustration to better understand the early mechanisms underlying the development of externalizing behavior.

Role of Parental Affective Dysregulation

According to Crockenberg et al. (2008), maternal behavior plays a key role in the development of a child's attention behavior and externalizing trajectories. One of the most well-documented risk factors during infancy of current and later externalizing problems is maternal depression (Campbell, Matestic, von Stauffenberg, Mohan, & Kirchner, 2007; Downey & Coyne, 1990). There is an extensive body of evidence to suggest that caregiver depression interferes with the ability to sensitively parent (Cicchetti, Rogosch, & Toth, 1998). Similarly, maternal anxiety has been shown to relate to problem behaviors in children, including anxiety/depression, attention deficit disorders, and oppositional defiant disorders (Meadows, McLanahan, & Brooks-Gunn, 2007). Thus, maternal affective dysregulation during infancy is commonly viewed as one of the strongest environmental predictors of child problems. The adoption design provides a unique opportunity to examine whether associations between caregiver affective state and child externalizing problems are found when family members are genetically unrelated.

Current Study

In the present study, a prospective adoption design is employed to examine hypotheses regarding the environmental moderation of genetic risk for externalizing behavior. In this design, the infant adopted by nonrelative parents shares 50% of his/her segregating genes with each of the birth parents but none with the adoptive parents. Conversely, the adopted child shares 100% of the rearing environment with the adoptive parents but none of the rearing environment with the birth parents. As such, genetic influences can be inferred based on similarities between the child and the birth parent(s), and environmental influences can be inferred based on similarities between the child and the adoptive parents. This design strength results from the fact that children placed in nonrelative adoptive homes do not share genes with their caregivers; thus, genetic influences cannot account for associations often seen between the caregiving environment and child behavior.

To date, only one other prospective longitudinal adoption study has examined genetic and environmental effects on social processes during infancy: the Colorado Adoption Project (CAP; Plomin, DeFries, & Fulker, 1988). Although the CAP has yielded evidence of GxE interactions on social behaviors during later childhood (Hershberger, 1994; O'Connor, Caspi, DeFries, & Plomin, 2003), GxE interaction effects during infancy have not been published to date. In the present study, we built upon the methods of the CAP by collecting observational assessments of social processes in the adoptive family and by using a fine-tuned assessment protocol with birth parents that included multiple measures of each targeted social construct to increase the reliability of genetic indices. By using a theory- and research-guided approach to carefully match birth parents' social characteristics with hypothesized externalizing and internalizing child outcomes a priori (Leve et al., 2007), we sought to increase the likelihood that GxE interactions would be detected in those domains.

Guided by the research reviewed above, we formulated three hypotheses: (a) that genetic effects on pathways to externalizing behavior could be traced intergenerationally via the association between birth parent externalizing behavior and heightened infant attention to frustration; (b) that adoptive parent affective state (e.g., anxious or depressive symptoms) would relate to infant attention to frustration, with higher levels of anxiety and depression being associated with greater infant attention to frustration; and (c) that the affective state of the adoptive parents would moderate genetic influences on infant attention during a frustration task, increasing the association between birth parent externalizing problems and infant attention to frustration.

Because substance use during pregnancy can confound genetic effects on development, we examined birth mother prenatal substance use in a secondary set of analyses.

Methods

Participants

The sample consisted of participants in the Early Growth and Development Study (EGDS), an ongoing, longitudinal, multisite study of 361 linked triads of adopted children, adoptive parents, and birth parents. The primary goal of the EGDS is to examine the effects of genotype–environment interaction and correlation on the social and emotional development of young children. The recruitment of participants occurred between 2003 and 2006, beginning with the recruitment of adoption agencies ($N = 33$ agencies in 10 states located in the Northwest, Mid-Atlantic, and Southwest regions of the United States). The participating agencies reflected the full range of adoption agencies in the United States: public, private, religious, secular, those favoring open adoptions, and those favoring closed adoptions. Each adoption agency appointed a liaison from their organization to assist with recruitment. The agency liaisons identified participants who completed an adoption plan through their agency and met the study’s eligibility criteria: (a) the adoption placement was domestic, (b) the baby was placed within 3 months postpartum, (c) the baby was placed with a nonrelative adoptive family, (d) the baby had no known major medical conditions such as extreme prematurity or extensive medical surgeries, and (e) the birth and adoptive parents were able to read or understand English at the eighth-grade level. The study participants were representative of the adoptive and birth parent populations that completed adoption plans at the participating agencies during the same time period (Leve et al., 2007).

The analytical sample for this report consisted of 348 linked triads from the first wave of assessment from whom at least two of the three members of the triad had data on the variables used in the present study. In addition, data from 105 birth fathers (BF) are included. The children were 9 months old during the assessment, $M = 9.20$ ($SD = .55$ months). Four triads were excluded from subsequent analyses on the basis of infant age (i.e., 1 child was 6 months old, and 3 children were more than 12 months old). Forty-three percent of the children were female. The mean child age at the adoption placement was 3 days ($SD = 5$ days). The adoptive families were typically college-educated, middle-class families, and the mean ages of adoptive mothers and fathers were 37 and 38, respectively. The adoptive parents had been married an average of 11.8 years ($SD = 5.1$ years), and 51% of the adoptive families had at least one additional child in the home at the time of the writing of this report. Information about which adoptive parent was the primary caregiver was not collected. The birth parents typically had high school or trade school education levels and household incomes under \$25,000, and the mean ages of birth mothers and fathers was 24 and 25, respectively. There were no significant differences in demographic characteristics between the full EGDS sample and the participants in the analytical sample used in this report. Complete demographic information on the full EGDS sample regarding adoptive mother (AM), adoptive father (AF), birth mother (BM), and birth father (BF) age, race, education, and income is presented in Leve, Neiderhiser, Scaramella, and Reiss (2008).

The study design included a birth parent in-person assessment between 3- and 6-months postpartum and an adoptive family in-person assessment when the child was 9 months old. The birth parent assessment was 2 h long and was conducted in the home or in another convenient location; the adoptive family assessment was 2-½ h long and was conducted in the home. The participants were paid for their time. For the birth and adoptive parent assessments, the interviewers asked computer-assisted interview questions, and each participant independently completed a set of questionnaires. In addition, the adoptive parents were observed individually (and once conjointly) in a series of interaction tasks with their child. The

birth and adoptive parent assessments were completed by separate teams of interviewers to prevent data sharing across members of the adoption triad. The interviewers completed a minimum of 40 hours of training, including a 2-day group session, pilot interviews, and videotaped feedback prior to administering interviews with study participants. All interviews were audio- or video-recorded, and feedback was provided by a trained evaluator for a random selection of 15% of the interviews to ensure adherence to the study's standardized interview protocols. Additional details on the EGDS study recruitment procedures, sample, and assessment methods can be found in Leve et al. (2007).

Measures

Genetic Risk for Externalizing Behavior

To estimate genetic risk for externalizing behavior, we administered three birth parent self-report measures considered to be indicators of externalizing behavior: lifetime alcohol, tobacco, marijuana, and other drug (ATOD) dependence; delinquency; and novelty seeking.

ATOD dependence—ATOD dependence was measured using a modified version of the Composite International Diagnostic Interview-Short Form (CIDI-SF) Alcohol and Drug Dependence scales (Kessler, Andrews, Mroczek, Ustun, & Wittchen, 1998). The original CIDI-SF questions were modified for this study to pertain to lifetime use (rather than 12-month use) and to include a set of tobacco dependence questions. The indicators of lifetime problem use of alcohol, tobacco, marijuana, and other drugs were then combined to form a composite ATOD dependence score. Alcohol dependence was calculated as the sum of seven items from the CIDI-SF assessing alcohol dependence symptoms in the past 12 months ($BM \alpha = .82$, $BF \alpha = .85$). For tobacco dependence, a 4-point scale was created using questions regarding past or current regular tobacco use and two dependence symptoms: 0 (*never used*), 1 (*past/current regular use but other dependence symptoms denied*), 2 (*one dependence symptom endorsed*), 3 (*both dependence symptoms endorsed*). Marijuana dependence was calculated as the sum of seven CIDI-SF dependence items ($BM \alpha = .85$, $BF \alpha = .88$). Other drug dependence included the following eight substance classes: sedatives, tranquilizers, amphetamines, painkillers, inhalants, cocaine, heroin, and hallucinogens. More than half of the birth parents reported use of one or more of these substance classes. However, use within any specific substance class was relatively low (14–26%). Therefore, we created dependence symptom scales for each substance class (based on the sum of CIDI-SF dependence items for that substance class; $BM \alpha = .63$ – $.97$, $BF \alpha = .74$ – $.96$). The other drug dependence indicator was calculated by averaging the standardized dependence symptom scales across each substance class ($BM \alpha = .85$, $BF \alpha = .83$).

The tobacco, alcohol, marijuana, and other drug dependence indicators were each standardized, and a composite ATOD dependence score was created ($BM \alpha = .72$, $BF \alpha = .71$).

Delinquency—Delinquency was measured using the 38-item Elliot Social Behavior Questionnaire (Elliot, Huizinga, & Ageton, 1985). On this self-report delinquency scale, birth parents rated items reflecting their engagement in various delinquent behaviors (e.g., purposely damaged or destroyed property, purposely set a fire, carried a hidden weapon, or stole something). Items were summed to create a delinquency score ($BM \alpha = .88$, $BF \alpha = .91$). The scales were log-transformed to reduce skewness.

Novelty seeking—Novelty seeking was based on the 20-item novelty-seeking subscale of the Temperament Character Inventory ($BM \alpha = .73$, $BF \alpha = .68$; Cloninger, Svaric, & Przybeck, 1993). This measure of general temperament and character is composed of 125 true/false items. Higher scores on the novelty-seeking subscale indicate an exploratory and novelty seeking

personality, impulsive decision-making, quick loss of temper, and excitable, enthusiastic, easily bored, and ardent behavior.

Associations among the externalizing indicators—For the BMs, the three standardized indicators (ATOD dependence, delinquency, and novelty seeking) were significantly correlated ($r = .28$ to $.41$, $p < .001$) and each loaded significantly on a one-factor solution (.76, .79, and .69, respectively), forming an internally consistent scale ($\alpha = .61$). The BF indicators did not show the same level of internal consistency, with a nonsignificant correlation among one set of variables and a marginally internally consistent scale ($\alpha = .56$). Still, the three indicators all loaded significantly on a one-factor solution (.73, .85, and .59, respectively). The three BM externalizing behavior indicators were used in the SEM, and a factor score was created for use in graphing. A BF externalizing behavior factor score based on the 99 BFs with data on all three indicators was calculated and used in the BF analyses.

Adoptive Parent Affective State

Adoptive parent affective state was examined using the Beck Anxiety Inventory (BAI; Beck & Steer, 1993a) and the Beck Depression Inventory (BDI; Beck & Steer, 1993b). Anxiety symptoms were calculated as the sum on the 21-item BAI (AM $\alpha = .76$, AF $\alpha = .74$). Depressive symptoms were calculated as the sum of 20 items from the 21-item BDI (AM $\alpha = .72$, AF $\alpha = .75$); the suicidal ideation item was not administered in the present study to minimize situations where clinical follow-up would be necessitated. The Anxiety and Depressive scales were log-transformed to reduce skewness. The resulting scales were correlated for AMs ($r = .48$) and AFs ($r = .54$) but were not correlated between parents. Thus, we standardized the Anxiety and Depression scales for each parent based on the means. Higher scores indicated greater anxious/depressive symptoms.

Infant Attention During a Frustration Task

Infant attention to frustration was measured during a barrier task as part of the Laboratory Temperament Assessment Battery assessment protocols (Goldsmith & Rothbart, 1996). In this video-recorded task, the child is seated in a high chair while six trials are run in 30-s intervals, alternating between neutral trials (trials 1, 3, and 5) and frustration trials (trials 2, 4, and 6). During each neutral trial, the same stimulating toy is presented on a high chair tray to the infant for 30 s. On each frustration trial, a clear Plexiglas barrier is placed between the infant and the toy for 30 s so that the infant can readily see the toy but cannot touch it. The barricade task was selected because of its developmental appropriateness and its similarity to attention to frustration tasks used in other studies during infancy (Crockenberg et al., 2008).

Infant attention during each trial of the barricade task was coded from video-recorded data using the Interest/Persistence scale from the Family Interaction Behavior Codes (Dogan et al., 2005). This scale assesses a child's level of engagement with the toy provided in the task. The primary measure is the amount of time the child engages with or attempts to engage with the toy. It also includes the amount of interest the child initially displays in the toy during each interval. A child scoring high on interest shows attention to, focus on, and thorough attempts to explore the toy provided to him/her for an extended period of time. Trained coders rated infants on interest behavior during each of the six trials using a 9-point Likert-type scale: 1 (*not at all characteristic*) to 9 (*mainly characteristic*). Thirty-five percent of the infants were dual-coded by independent coders to establish reliability. Coder intraclass correlations ranged from .91 to .97 across the six trials, indicating high inter-rater reliability.

A factor analysis with oblique rotation supported a two-factor solution and a distinction between the trial types; neutral trials loaded .76–.84 on one factor, while frustration trials loaded .77–.86 on a second factor (r between factors = .315, $p < .001$). Attention ratings were

standardized at each trial. The measure of attention during neutral trials was calculated as the mean of standardized ratings on trials 1, 3, and 5 ($\alpha = .71$), and attention during frustration trials was calculated as the mean on trials 2, 4, and 6 ($\alpha = .76$). Attention during the frustration trials served as the dependent measure of interest for the current analyses; attention during the neutral trials was included as a control variable to take into account individual differences in general attention levels during neutral situations.

Additional Control Variables

Several additional control variables were included to control for phenomena noncentral to the study hypotheses.

Openness in adoption—To control for similarities between birth and adoptive families resulting from contact and knowledge between parties, we controlled for the level of openness in the adoption. We measured openness in adoption using a composite index of BM, BF, AM, and AF perceived openness using a 7-point scale: 1 (*very closed*) to 7 (*very open*). Interrater agreement was high (r range = .66–.81; Ge et al., 2008).

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

BM prenatal ATOD use—Maternal prenatal ATOD use can confound estimates of the genetic influences related to substance use (such as our ATOD dependence measure, described above). Thus, prenatal ATOD use was included as a control variable in a secondary set of analyses. The BMs reported prenatal ATOD use on each of ten substance classes (tobacco, alcohol, sedatives, tranquilizers, amphetamines, painkillers, inhalants, cocaine, heroin, and hallucinogens) using a pregnancy history calendar method developed for the study. The BMs were asked to identify key events throughout the pregnancy period (e.g., birthdays, holidays, and vacations) and to note them on a 1-page life history calendar, which has been shown to be an effective and reliable tool for recording retrospective events and conditions (Caspi, Moffitt, Thornton, et al., 1996). The BMs then responded to questions about their substance use during the pregnancy period, using the calendar as a reference to facilitate recall.

For tobacco use, the BMs reported whether they had smoked cigarettes in the 1st, 2nd, and/or 3rd trimesters of this pregnancy using a yes/no format; 59.6% reported not smoking, and 26.2% reported smoking during all three trimesters. For the remaining substances classes, we used a 5-point scale for the 9-month pregnancy period: 0 (*never*) to 4 (*regularly/most days or every day*). Few BMs reported more than rare alcohol use (5.4%) or any level of use within the other drug classes (ranging from 0.3% for hallucinogens to 16.4% for marijuana), but 27.0% of the mothers reported use of at least one of these substances. Given the skewed distributions of the use indicators, we considered a variety of rescaling options when constructing an overall scale. Dichotomizing all 10 indicators—0 (*no use*) or 1 (*any use*)—yielded an internally consistent scale, Cronbach's α (KR-20) = .67. Hallucinogen use was omitted during scale development due to low frequency but was included in the final scale because of face validity. The sum of dichotomous indicators was positively skewed and was therefore collapsed into a 5-point scale: 0 (*prenatal use of no substances*) to 4 (*prenatal use of four or more substances*).

Data Analysis Approach

The analyses were aimed at examining genetic, family environment, and Genotype x Family Environment effects on infant heightened attention during the frustration task. Significant correlations between an infant characteristic and a birth parent characteristic belonging to the same underlying construct were inferred to reflect genetic influences.

SEM using Mplus (Muthén & Muthén, 2006) was used to test whether BM externalizing behavior (a latent factor indexed by multiple indicators), AM and AF affective state (observed variables), and the interaction between them predicted infants' attention during the frustration trials (an observed variable). We tested the interactions involving AM and AF affective state separately, while controlling for the other adoptive parent's state (e.g., BM Externalizing x AM Affective State, controlling for AF affective state). Three control variables were included in the models: infant sex, openness in adoption, and infant attention during the neutral trials. In addition, we examined BM substance use during pregnancy in follow-up analyses to determine whether genetic influences were confounded by prenatal ATOD use.

All of the variables were standardized prior to modeling and used the maximum likelihood algorithm with robust standard errors (MLR) to derive parameter estimates in the presence of missing data. Given the lack of established fit statistics for models involving statistical interaction with latent variables, we followed a series of steps to test these models. First, to test whether the model provided an adequate fit without the interaction term, we computed an MLR chi-square (asymptotically equivalent to Yuan-Bentler T2 χ^2 ; Muthén & Muthén, 2006) and examined RMSEA and CFI fit indices. Second, we used the Satorra-Bentler log-likelihood difference chi-square test to compare a model with the path from the interaction term to the outcome to the model without the path, as one is mathematically nested within the other. Third, when model fit was improved and an individual interaction term was significant, we examined the direction of effects by splitting the sample at the mean of the moderating variable and examining the effects of BM externalizing problems on infant attention to frustration within the resulting subsamples. We used the mean-split approach rather than splitting the sample 1 *SD* above and below the mean due to the small sample sizes that resulted in the latter approach. Nonetheless, we ran preliminary follow-up analyses splitting the sample at 1 *SD* above and below the mean and at 0.5 *SD* above and below the mean to examine comparability of the results using the different approaches.

The same set of interactions was also tested using the reduced BF subsample. We could not use a latent BF externalizing problem variable in the SEM due to the smaller sample size and relatively low association between two of the three ATOD use variables. Thus, there was no advantage to testing the hypotheses in this subsample using SEM. Instead, we used linear regression analyses in SPSS. We examined a BF externalizing problem factor score (list-wise $n = 99$), AM affective state, and the interaction between these two variables as predictors of infant attention during the frustration trials. A parallel regression model in which BF externalizing problems interacted with AF affective state was considered separately.

Results

We first examined the means, standard deviations, and bivariate correlations among study variables. Next, we performed SEM to test the hypothesis that BM characteristics would interact with adoptive parent affective state to predict infant heightened attention during the frustration task. As a subsidiary analysis, we examined this hypothesis using a regression approach with the BF data. Finally, we explored prenatal substance use effects as a potential mediating pathway.

Descriptive Statistics and Correlations

The analytic sample consisted of 348 linked triads for whom data were available on at least two of the three members of the triad. Means, standard deviations, correlations among study variables, and n sizes for each variable are summarized in Table 1. Infant attention during the frustration trials was moderately associated with infant attention during the neutral trials ($r = .33, p < .001$). The correlation matrix also indicated significant associations among the three BM externalizing measures, significant associations between two of the three BF externalizing

measures, and significant associations among about half of the BM–BF externalizing behavior cross correlations. In addition, there were several modest but significant correlations among the remaining variables. For example, BM prenatal ATOD use was correlated with the BM externalizing measures and with two of the BF externalizing measures. BM delinquency was also modestly associated with attention during the frustration trials ($r = .13, p < .05$) and was inversely associated with AF affective state ($r = -.12, p < .05$), and AM affective state was associated with openness ($r = .12, p < .05$). Otherwise, the study variables were no more associated than would be expected by chance.

Evaluation of BM x Adoptive Parent Effects

The SEM analyses used full information maximum likelihood, which has been shown to provide unbiased estimates when data are missing at random (Arbuckle, 1996) and to offer greater statistical efficiency compared to mean-imputation, list-wise, and pair-wise deletion methods (Wothke, 2000). The first series of models tested infant attention during the frustration trials regressed on the latent BM externalizing behavior variable (ATOD dependence, delinquency, and novelty seeking), AM affective state, and their interaction; AF affective state, adoption openness, infant attention during neutral trials, and infant sex were statistically controlled.

The first of these models (prior to entry of the interaction term) showed an acceptable fit to the data. Higher levels of AM anxious/depressive symptoms were modestly associated with higher levels of infant attention during the frustration trials ($p < .05$). Higher levels of BM externalizing behavior indicated a trend toward being significantly associated with higher levels of infant attention during the frustration trials ($p < .10$). Other than infant attention during the neutral trials ($p < .001$), none of the control variables were associated with infant attention to frustration in this model. Next, a model in which the interaction between BM externalizing problems and AM affective state was fitted to the data. The results are summarized in Figure 1. Compared to the first model, in which this path was fixed at zero, this model fit the data significantly better, Satorra-Bentler χ^2 difference ($df = 1$) = 6.67, $p < .05$. More importantly, the interaction term also was significant ($p < .05$). The AM affective state remained significant ($\beta = .10, p < .05$), and the BM externalizing behavior path became significant in the interaction model ($\beta = .17, p < .05$).

Decomposition of Interaction Between BM Externalizing Behavior and AM Affective State

To examine the nature of this interaction, the model was run within the subsamples that were above and below the mean on the AM affective state variable; nonsignificant covariates (AF affective state, infant sex, and adoption openness) were omitted given the reduced subsample sizes. In the subsample above the mean on the AM affective state variable ($n = 184$), the model provided a good fit to the data, $\chi^2 = 5.17, df = 6, p = .523$; CFI = 1.00; RMSEA = .00. The path from BM externalizing problems to the infant attention to frustration was significant ($\beta = .35, p < .01$). This model was then rerun with the subsample below the mean on the AM affective state variable ($n = 164$). This model also fit the data well, $\chi^2 = 6.45, df = 6, p = .38$; CFI = .99; RMSEA = .02. The path from BM externalizing problems to infant attention was not significant ($\beta = -.01$).

The interaction decomposition models were rerun to examine whether the pattern of effects remained when the sample was split at 1 *SD* above and below the mean and at 0.5 *SD* above and below the mean on the AM affective state variable. Due to the reduced sample sizes, these models were considered preliminary. The standardized path coefficients from BM externalizing to infant attention to frustration were in the same direction as the mean-split analyses for both models: .34 at 1 *SD* above the mean of AM affective state ($n = 62$); .36 at 0.5 *SD* above the mean ($n = 121, p < .05$); $-.04$ at 1 *SD* below the mean ($n = 68$); and $-.02$ at

0.5 *SD* below the mean ($n = 121$). These follow-up analyses support the effects found with the mean-split approach. Infant attention to frustration was regressed on BM externalizing problem factor scores for the two mean-split groups (see Figure 2), reflecting the linear regression results using observed (versus latent) variables and list-wise deletion of cases with missing data.

Finally, we ran a similar series of models to evaluate whether AF affective state interacted with BM externalizing to predict infant attention during the frustration task. In these models, AM affective state and the other covariates (described above) were controlled. All main effect and interaction paths were nonsignificant, suggesting that the effects described above are specific to AM affective state.

Linear Regression: Evaluation of BF x Adoptive Parent Effects

The GxE hypotheses were examined in a parallel way with BF data using linear regression techniques due to the nonconvergence of the latent factor and the smaller sample size. Externalizing behavior problems were represented by the factor score composed of BF ATOD dependence, delinquency, and novelty-seeking indicators. Infant attention during the frustration trials was not predicted by this BF composite alone or in interaction with the AM affective state variable, $F(3, 81) = .587$, *ns*, or the AF affective state variable, $F(3, 78) = .255$, *ns*.

Exploratory Models Controlling for BM Prenatal ATOD Use

Next, exploratory models were run to test whether the effect of the interaction between BM externalizing problems and AM affective state on infants' attention during the frustration trials was better accounted for by BM prenatal ATOD use. In general, all models and path coefficients were regarded with caution in these analyses due to the functional relationship between general and prenatal ATOD use (i.e., mothers who report never using will also report never using during pregnancy, and mothers who use during pregnancy typically use outside of pregnancy).

BM prenatal ATOD use was entered in the full model as a mediator of interaction effects. That is, BM prenatal ATOD use was regressed on BM externalizing problems, AM affective state, and their interaction, and infant attention to frustration was regressed on BM prenatal ATOD use, attention during neutral trials, infant sex, AF affective state, and adoption openness but not directly on terms involved in the interaction. BM externalizing was significantly associated with BM prenatal ATOD use, but the interaction term was not significant. Additionally, BM prenatal ATOD use did not significantly predict infant attention to frustration. This model was compared with a model that allowed direct predictive paths from BM externalizing, AM affective state, and their interaction to infant attention during the frustration trials. The latter model fit the data better, Satorra-Bentler χ^2 difference ($df = 3$) = 15.46, $p < .05$, and the path from the interaction term to the infant attention outcomes was significant ($p < .01$). Similarly, within the subsample that was above the mean on AM affective state, the reduced model (i.e., exclusion of infant sex, AF affective state, and openness in adoption) provided a marginal fit to the data, $\chi^2 = 14.924$, $df = 9$, $p = .093$; CFI = .960; RMSEA = .060. BM externalizing problems predicted BM prenatal ATOD use ($\beta = .72$, $p < .001$) and infant attention during the frustration trials ($\beta = .48$, $p < .05$), whereas BM prenatal ATOD use did not predict infant attention during the frustration trials ($\beta = -.19$, $p = ns$). The model that fixed the direct path from BM externalizing problems to infant attention during the frustration trials did not fit the data adequately, $\chi^2 = 25.381$, $df = 10$, $p = .005$; CFI = .896; RMSEA = .091. Therefore, the findings did not support BM prenatal ATOD use as an explanation for the GxE findings described above.

Discussion

Recently, researchers from developmental psychology and behavioral genetic perspectives have called for and highlighted the potential value of integrating behavioral genetic and developmental research to improve outcomes for youth and families (Reiss & Leve, 2007; Rutter, 2005). In the current study, we made a preliminary effort to leverage the unique opportunity provided by an adoption design to understand how a specific family environmental experience—infant exposure to adoptive parent anxious/depressive symptoms—might moderate a genetic propensity for externalizing behavior very early in development and, as such, increase the understanding of causal mechanisms during early childhood.

The results suggest that infants are more likely to show heightened attention during a frustration task when they are exposed to an early rearing environment involving elevated maternal anxious/depressive symptoms. Although the direct association between maternal anxious/depressive symptoms and infant attention to frustration was modest in this study, this main effect finding is consistent with prior findings that demonstrate the significant effects of maternal depression in early childhood on young children's problem behaviors (Downey & Coyne, 1990). However, it is unique in that the parents and children were genetically unrelated in the present study, thereby suggesting a pure environmental mechanism of transmission that is unclouded by genetic factors. Beyond the direct effects of AM affective state, the SEM results suggest that maternal affective state moderates the effects of genetic risk for externalizing behavior. That is, the direct effect of birth parent externalizing behavior on infant attention to frustration was amplified in the context of elevated levels of adoptive mothers' anxious/depressive symptoms. Researchers have examined the moderating role of *genetic* risk for psychopathology (e.g., birth parent psychopathology; Riggins-Caspers, Cadoret, Knutson, & Langbehn, 2001), but, to our knowledge, this study is the first attempt to examine the affective state of rearing parents on moderating genetic influences in young children. As is shown in Figure 2, genetic risk for externalizing behavior was unrelated to infant attention when AMs were below the mean on anxious/depressive symptoms. Conversely, when AMs were above the mean, genetic risk was exacerbated, with the association between BM externalizing behavior and infant attention during a frustration task becoming significant ($\beta = .35, p < .01$).

The specific phenotype measured in the present study was infants' attention, interest, and persistence to a frustrating task. As such, this behavior was considered to reflect an inability to direct attention away from a stressful situation (rather than reflecting more general attention processes). The follow-up analyses examining whether the same GxE processes operated on infant attention during the neutral trials yielded nonsignificant results, suggesting the specificity of maternal affect in moderating effects to situations where the infant might feel frustrated and lack autonomy. Through a combination of genetic liability and emotionally unavailable and dysregulated maternal caregivers, these infants might begin to react with increased attention and intensity under conditions of frustration, perhaps in an effort to solicit caregiver attention or involvement from an anxious or depressed mother. Such reactions during infancy might be the forerunners of noncompliance and reactivity during toddlerhood, when children are in similarly frustrating situations and are subjected to constraints placed upon them by their caregivers (e.g., when a parent asks a child to clean up his/her toys). The reactions of some children can increase under such conditions, escalating into coercive cycles with the parent during childhood (Shaw et al., 1994, 1998) and evolving into delinquency and antisocial behavior that continues into adolescence (Patterson, Reid, & Dishion, 1992).

Parental Sex Differences

Although support was found for direct and moderating effects of AM affective state on infant attention during a frustration task, the AF results did not support the hypotheses. Similarly, the GxE interaction was not significant when the BF data were modeled. Together, these

nonsignificant results for fathers suggest a pattern of maternal linkages to externalizing behavior in young children, in terms of both the rearing environment and genetic influences. The lack of significant findings for the unique contributions of AF affective state was not surprising; prior work has found stronger associations between maternal affective state and child adjustment than between paternal affective state and child adjustment (Meadows et al., 2007). Measuring the respective caregiving roles of both parents in future studies would help elucidate the extent to which this rearing parent environmental effect results from differential levels of caregiving between mothers and fathers or whether there is something unique about maternal affective state irrespective of the level of her caregiving. Alternatively, given that the parenting factors used in the present study have been validated primarily with samples of mothers, other aspects of paternal caregiving in early or later development (e.g., facilitating problem solving in middle childhood) might play a more important role in the development of child problem behavior.

The lack of significant BF effects was unexpected and suggests the possibility of sex-linked transmission of genetic risk for externalizing behavior. Although this is the largest BF sample in a single prospective adoption design collected to date, we could not test models that aggregate genetic risk by forming a composite between BM and BF data because of the large number of nonparticipating BFs and the lack of convergence of indicators for the participating BFs. Further, the results from the BF regression analyses indicate that the direction of effects does not resemble that of BM, suggesting that a larger sample would not alter the pattern of findings. Sex differences in the heritability of externalizing behavior have been examined in a number of studies. Although some researchers have reported similar patterns of heritability across the sexes (Rhee & Waldman, 2002), others have found that genetic influences are significantly higher in females than in males. For example, Tuvblad, Grann, and Lichtenstein (2006) found heritability estimates of 6% for adolescent boys' antisocial behavior and 59% for adolescent girls' antisocial behavior.

A polygenetic multiple threshold model can be used as a framework to consider the sex differences found in this study (Rhee & Waldman, 2002). In such a model, the sex less afflicted with a behavior might require a greater liability to express that behavior. In the case of externalizing behaviors, females are the less affected sex; thus, the relatives of affected females might be at greater risk of developing externalizing problems than relatives of affected males. In the present analyses, the associations between BM externalizing behavior (compared to BF externalizing behavior) and infant attention during a frustration task were more pronounced, thereby permitting sufficient variation to allow for the detection of environmental moderation of this genetic effect. In addition, as is shown in Table 1, the mean levels of externalizing behavior were only modestly lower for BMs than for BFs, an atypical finding for adult samples that provides some corroborating evidence for the polygenetic multiple threshold model. Parallel evidence in support of polygenetic multiple threshold processes comes from clinical research involving a gender paradox in the severity of disorders. For disorders with an unequal sex ratio, individuals with the lower prevalence rates tend to be more seriously affected and to have more co-occurring diagnoses (Loeber & Keenan, 1994). For example, the likelihood of girls with attention deficit disorder to develop conduct disorder is 40 times greater than for girls without attention deficit disorder, compared to a factor of 14.7 for boys (Szatmari, Boyle, & Offord, 1989). Although the lack of significant effects for the BF analyses should be interpreted with caution because they were counter to study predictions, the two lines of research described above suggest that future research should consider sex-linked explanations when examining GxE interaction processes on externalizing behaviors in early childhood.

Future Directions: Implications for the Development of Preventive Interventions

In conducting this study, we had an implicit goal of exploring possibilities for the development of future preventive interventions by capitalizing on the EGDS adoption study design, which provides information about the processes whereby early environmental experiences can offset or exacerbate genetic strengths and liabilities. As has been noted by others, adoption is an effective intervention, particularly for children living in suboptimal environments who are placed in more enriched environments within the first few months of life (Rutter, O'Connor, & the English and Romanian Adoptees Study Team, 2004; van IJzendoorn & Juffer, 2006). Children adopted at an early age typically fare better than their nonadopted counterparts on a range of cognitive and social outcomes. However, in addition to mean level shifts in behavior resulting from adoption, the full adoption design permits the examination of the mechanisms whereby differing early environmental experiences can lead to healthy or less optimal adjustment. In the present study, AM anxious/depressive symptoms moderated the effect of genetic risk for early externalizing problems. If this effect were to be replicated and extended in future research, preventive interventions targeting mothers with affective problems might improve the mental health of the caregiver, in turn alleviating exposure to adverse environmental conditions that might offset the expression of children's genetic risk for externalizing behavior. Although this possibility cannot be directly addressed in the present study until the link between infant frustration and later externalizing problems is confirmed, it is tentatively proposed as a hypothesis for further investigation. Notwithstanding this caveat, the results from two recent intervention trials have provided the first evidence that interventions aimed at reducing parental depression might significantly reduce child behavior problems (Weissman et al., 2006; Shaw, Dishion, Connell, Wilson, & Gardner, in press), thereby supporting this hypothesis.

Alternative Explanations

Although the results of the present analyses are unique and appear to be robust, several alternative explanations for AM environmental moderation of genetic effects could be possible. First, intrauterine exposure to toxins might mask or confound genetic effects. Although BM prenatal ATOD use does not appear to be a causal mechanism in the associations found here, additional prenatal experiences (e.g., exposure to environmental toxins) have not been examined; thus, other prenatal factors could be associated with the pattern of findings described here.

Second, trends in adoption practices such as selective placement (agency matching of birth and adoptive parent characteristics) and openness (contact and knowledge between birth and adoptive families) can pose a threat to the assumptions underlying the adoption design and bias model estimates. For example, an adopted child might be more likely to resemble his/her birth parents (artificially inflating genetic estimates) if the birth parents are in direct contact with the child or if the birth parents have actively selected an adoptive home that closely mirrors their own environment. As such, an openness construct was included in the analytical models; this construct neither yielded a significant association with the dependent measure nor accounted for significant variance in the relationships between BM externalizing behavior, AM affective state, and infant attention during a frustration task (see Figure 1). Ruling out the possible effects of selective placement is more complex because the associations between a birth parent characteristic and an adoptive parent characteristic could indicate selective placement or could indicate genetically influenced evocative behaviors in the child that have modified adoptive parent behavior to be more similar to birth parent characteristics. Previously, we correlated a set of birth parent characteristics with a set of adoptive family characteristics that were *unlikely* to be influenced by evocative effects (i.e., demographic characteristics) to consider the possible effects of selective placement (Leve et al., 2007). No relationship was

significant. Thus, systematic selective placement was not identified, although it cannot be fully ruled out as a possible explanation for the present effects.

Third, the GxE effects found in the present study could have resulted from evocative genotype–environment correlation (rGE), which has been found in other samples of infants and toddlers (Forget-Dubois et al., 2006). In the present study, evocative rGE would have been noted if the genetically influenced externalizing behavior of the infant directly influenced AM anxious/depressive symptoms and confounded the GxE effects. Although longitudinal designs are needed to truly separate the directionality of evocative effects, the correlation between AM affective state and BM externalizing behavior in this study was nonsignificant. Similarly, the correlations between fathers (adoptive and birth) were nonsignificant (see Table 1). These results suggest that rGE is likely not confounding the moderating effects shown by AM affective state.

Fourth, in the present study, we examined a single outcome. Based on the research reviewed earlier, we hypothesized that attention, interest, and persistence toward a frustrating situation would reflect an underlying genetic predisposition toward later externalizing behavior, with children at genetic risk showing heightened attention when frustrating constraints were placed upon them. Alternatively, the barrier frustration task might simply elicit vulnerabilities to negative affectivity rather than differences in attention processes. To test this alternative hypothesis, we examined the final models using negative affect during the barrier task as the outcome using the same coding system as the attention variable. No significant GxE effects were found. Further, there were significantly stronger associations between negative affect and attention in the control trials ($r = -.53, p < .001$) than in the frustration trials ($r = -.15, p < .05$), suggesting the relative independence of attention and negative affect within the context of a frustrating situation.

Fifth, based on prior findings in the literature, we hypothesized that the primary genetic risk factor associated with infant attention would be externalizing problems and that the primary early environmental risk factor would be adoptive parent anxious/depressive symptoms. However, given the modest amount of variance accounted for in our results, it is plausible that additional genetic influences and family environmental contexts affect infant attention to frustration. Although rates of adoptive parent externalizing behavior were low in this sample, we tested whether similar main effects and interaction effects would be found when adoptive parent antisocial behavior or novelty seeking was imputed as the environmental measure (using identical measures as were used for birth parents). In this post hoc set of analyses, no main effects or interaction effects were found, providing converging evidence for maternal affective state as a central environmental variable from birth to 9 months. We next examined alternative genetic risk factors and conducted a post hoc analysis of whether BM anxiety/depression (using identical measures as were used for adoptive parents) would show associations with infant attention during a frustration task. Although there was no main effect, the interaction between BM affective state and AM affective state was significant, suggesting a potential explanatory role of BM affective state. However, given the relatively high association between BM externalizing problems and BM affective state in this sample ($r = .40, p < .001$) and the fact that the original interaction between BM externalizing problems and AM affective state remained significant while controlling for BM affective state, the effects of BM affective state do not appear to be as strong as the effects of BM externalizing problems in this study. Further, the findings from prior research on attention to frustration have suggested associations with later externalizing problems (rather than internalizing problems), suggesting that our reliance on prior research to guide the selection of genetic and environmental risk variables in the present study was appropriate.

Limitations

To our knowledge, the present study is the first to examine how early exposure to maternal anxious/depressive symptom moderates genetic influences on risk for later externalizing behavior. The use of a prospective adoption design enabled us to make associations between the phenotypic behavior of the birth parent and the phenotypic behavior of an infant to examine underlying inherited predispositions toward externalizing behavior. However, the present study is not without limitations. First, because cross-sectional data were used, longitudinal pathways could not be tested. The longitudinal nature of the study will enable us to examine, in subsequent years, how the moderating effects of AM affective state unfold over time. Of relevance to informing preventive intervention, it will be critical to examine whether the mechanisms affecting infant attention are in fact found to lead the child toward a trajectory of early and persistent externalizing problems. Without such longitudinal data, we can only speculate about the intermediary phenotypic pathways to externalizing behavior and cannot make conclusive interpretations. Further, the moderating effects of the early home environment were already present at 9 months of age; studies beginning earlier in development are needed to help define the causal mechanisms whereby early exposure to maternal anxiety and depression result in the expression of risk in the child.

In addition, although findings from prior research on attention to frustration have suggested associations with later externalizing problems, it is likely that additional genetic influences and family environmental contexts affect infant attention to frustration. Specifically, attachment is a key environmental influence on later infant externalizing behavior (Smeekens et al., 2007), and cognitive functioning is known to be a heritable characteristic related to externalizing behavior (DeYoung et al., 2006). Such variables (not assessed at our 9-month assessment) might be associated with the infant attention to frustration variable. Additional research is needed to test these alternate environmental and genetic pathways.

Finally, although our sample was representative of birth and adoptive families from the agencies participating in this study, the adoptive families had high educational and economic backgrounds and low rates of externalizing problems compared to national norms. Thus, the discrepancy in sociodemographic characteristics often found between birth and adoptive parents was replicated in the present study, indicating the potential restriction of range in the environment that has been noted in prior reviews of the adoption design (Stoolmiller, 1999). However, in a recent systematic test of range restriction biases, McGue et al. (2007) found negligible effects on estimates of heritability and the environment, even when range restriction was present. Further, the adoptive parents in our study had an adequate range of depression and anxiety symptoms (Leve et al., 2007). Other researchers have found that genetic influences on externalizing behavior are more important in families with more advantaged environments, whereas the shared environment is more important in more economically disadvantaged environments (Tuvblad et al., 2006). Thus, the generalizability of findings to high-risk home environments and more ethnically diverse samples needs to be documented before stronger conclusions can be drawn.

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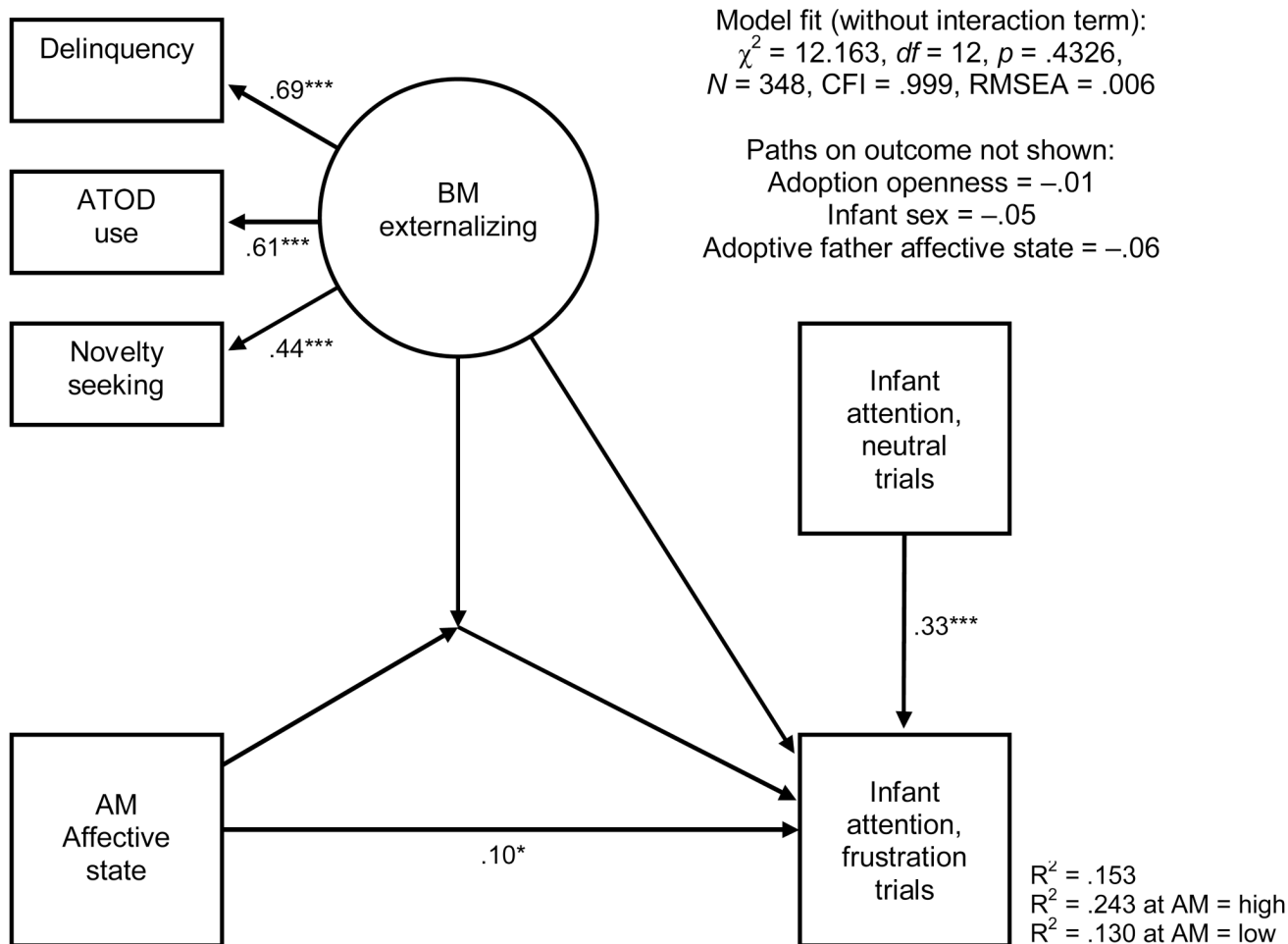


Figure 1. Standardized path coefficients from study variables to infants’ attention to frustration. *Note.* Also depicted are paths from birth mother (BM) externalizing behaviors and explained variance in outcome above and below the mean on the adoptive mother (AM) affective state variable (“AM = high” and “AM = low,” respectively). * $p < .05$. ** $p < .01$. *** $p < .001$.

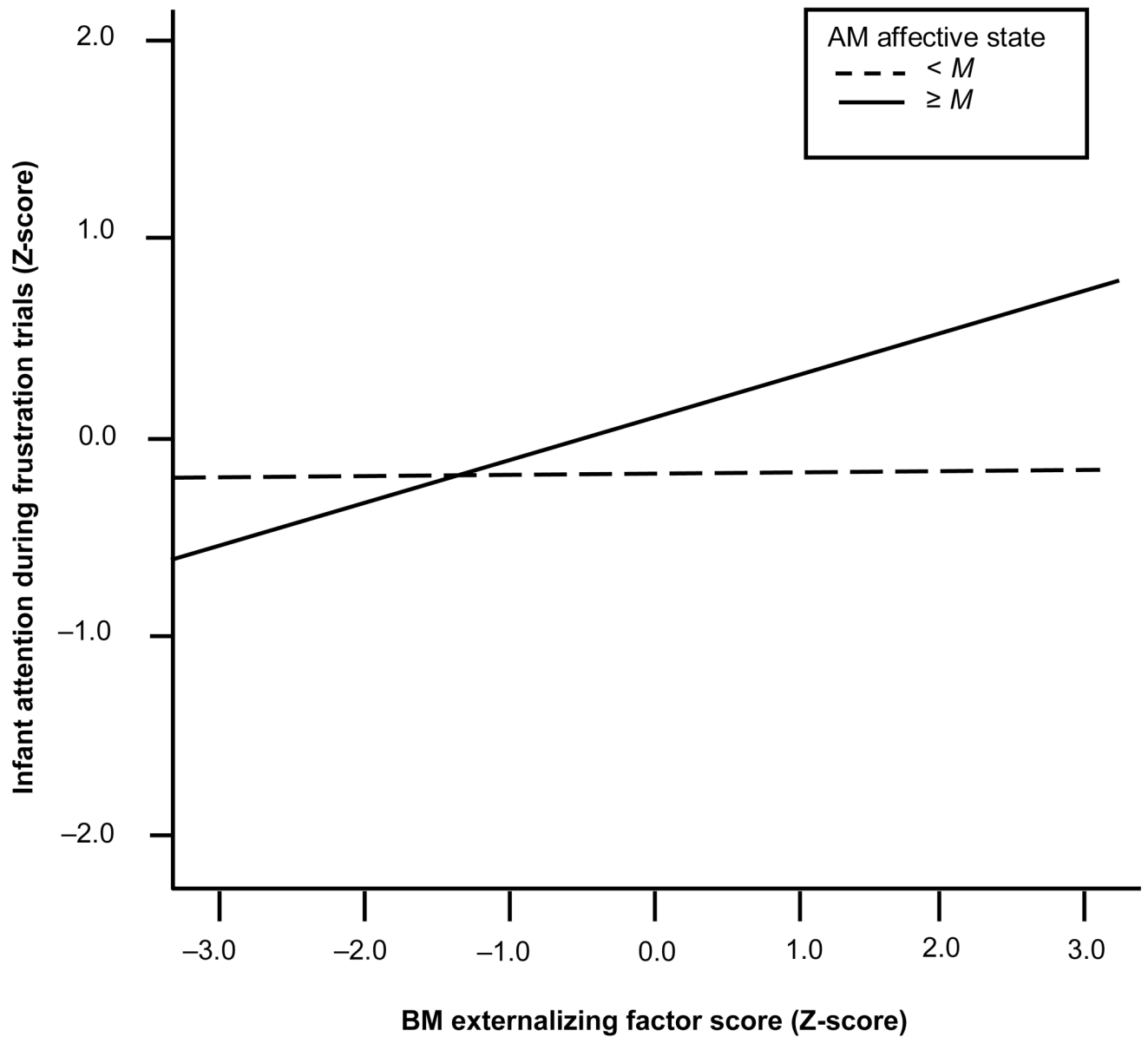


Figure 2. Relations between birth mother (BM) externalizing problems and infants' attention to frustration above and below the mean on the measure of adoptive mother (AM) affective state.

Table 1

Means, Standard Deviations, and Correlations Among Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12
1. Attention - frustration trials	—											
2. Attention - neutral trials	.33***	—										
3. Affective state (AF)	-.07	.01	—									
4. Affective state (AM)	.09	-.01	.05	—								
5. ATOD use (BF)	.09	-.05	-.09	.08	—							
6. Delinquency (BF)	.04	.08	-.06	-.06	.44***	—						
7. Novelty-seeking (BF)	.00	-.10	.13	-.12	.11	.33**	—					
8. ATOD use (BM)	.11	-.07	-.06	.01	.60***	.25*	-.11	—				
9. Delinquency (BM)	.13*	.01	-.12*	-.02	.33***	.27**	.08	.41***	—			
10. Novelty-seeking (BM)	.02	.04	-.03	.01	.15	.15	.06	.28***	.31***	—		
11. Prenatal ATOD use (BM)	.04	-.05	-.04	-.01	.52***	.23*	.01	.56***	.38***	.15**	—	
12. Openness in adoption	.03	.03	-.01	.12*	.02	.05	-.07	.10	-.01	.08	.08	—
<i>M</i>	3.04	5.23	0.00 ^a	0.00 ^a	0.00 ^b	47.28	12.24	0.00 ^b	41.93	10.57	1.01	4.74
<i>SD</i>	1.16	1.48	1.00	1.00	1.00	9.09	3.50	1.00	5.92	3.84	1.17	1.17
<i>N</i>	1	2	3	4	5	6	7	8	9	10	11	12
	312	314	336	348	103	105	104	338	340	340	347	348

Note. AF = adoptive father; AM = adoptive mother; BF = birth father; BM = birth mother; ATOD = alcohol, tobacco, and other drugs.

^a Mean of standardized Beck Anxiety Inventory (BAI) and modified Beck Depression Inventory (BDI) scores; unstandardized *M* (*SD*) BAI scores for adoptive fathers and mothers were 3.03 (3.09) and 3.84 (3.55), respectively; adoptive father and mother unstandardized *M* (*SD*) BDI scores were 2.89 (3.37) and 3.55 (3.11), respectively.

^b Composite score based on four standardized indicators.

* $p < .05$.

** $p < .01$.

*** $p < .001$.