Published in final edited form as:
J Am Acad Child Adolesc Psychiatry. 2009 November ; 48(11): 1102–1109. doi:10.1097/CHI.0b013e3181b8bfc0.

Structured Parenting of Toddlers at High versus Low Genetic Risk: Two Pathways to Child Problems

Leslie D. Leve, PhD, **Gordon T. Harold, PhD**, **Xiaojia Ge, PhD**, **Jenae M. Neiderhiser, PhD**, **Daniel Shaw, PhD**, **Laura V. Scaramella, PhD**, and **David Reiss, MD**

Abstract

Objective—Little is known about how parenting might offset genetic risk to prevent the onset of child problems during toddlerhood. We used a prospective adoption design to separate genetic and environmental influences and test whether associations between structured parenting and toddler behavior problems were conditioned by genetic risk for psychopathology.

Method—The sample included 290 linked sets of adoptive families and birth mothers and 95 linked birth fathers. Genetic risk was assessed via birth mother and birth father psychopathology (anxiety, depression, antisociality, and drug use). Structured parenting was assessed via microsocial coding of adoptive mothers' behavior during a clean-up task. Toddler behavior problems were assessed with the Child Behavior Checklist.

Results—Controlling for temperamental risk at 9 months, there was an interaction between birth mother psychopathology and adoptive mothers' parenting on toddler behavior problems at 18 months. The interaction indicated two pathways to child problems: structured parenting was beneficial for toddlers at high genetic risk but was related to behavior problems for toddlers at low genetic risk. This cross-over interaction pattern was replicated with birth father psychopathology as the index of genetic risk.

Conclusions—The effects of structured parenting on toddler behavior problems varied as a function of genetic risk. Children at genetic risk might benefit from parenting interventions during toddlerhood that enhance structured parenting.

Keywords

toddler; behavior problems; parenting; genetic influences; adoption design

Introduction

Understanding the etiological processes leading to early risk for psychopathology is critical to intervention efforts aimed at preventing the onset of psychiatric disorders. One of the strongest predictors of risk for psychopathology is inadequate parenting; numerous studies illustrate the role of parenting in the origins of psychiatric disorders.[1-2] In terms of discrete parenting behaviors that impact the development of psychopathology during early childhood, structured parenting (eg, providing clear instructions and structuring the child's time) has been shown prevent risk for problems, particularly when children are in social situations that demand compliance.[3-4] The benefits of structured parenting on reducing child problems has been shown across multiple contexts, including supermarket trips,[5] forbidden toy tasks,[6-7] and competitive games.[3] Further, randomized prevention trials have indicated that intervention effects on child

Corresponding Author: Leslie D. Leve, PhD, Oregon Social Learning Center, 10 Shelton McMurphey Boulevard, Eugene, OR 97401, USA (lesliel@oslc.org), telephone: 541-485-2711, fax: 541-485-7087..

psychopathology are mediated by parenting, with improvements in parenting predicting reductions in child disruptive behavior problems, highlighting the causal role of parenting behaviors on risk for psychopathology during early childhood.[8-9]

The purpose of the present study was to expand upon prior work by examining not only the main effects of parenting during toddlerhood, but additionally, to examine how structured parenting might offset genetic risk. A prospective adoption design was employed to separate genetic and environmental influences by linking early childhood behavior problems with psychopathology in birth parents and adoptive parenting behaviors, respectively. In order to control for behavioral risk occurring in infancy, two dimensions of infant temperament with known associations to later psychopathology and psychiatric diagnosis were incorporated: impulsivity and fearfulness.[1,10-13]

Recent studies suggest that additive models of parenting and child characteristics are insufficient for explaining the wide variation in child psychopathology. Rather, the effects of parenting on early childhood risk for psychopathology are often conditioned by child characteristics.[14-17] For example, negative parenting has been shown to be a stronger predictor of later externalizing symptoms and inhibition among children who have higher negative emotionality.[16] Similarly, child impulsivity has been shown to be most strongly related to later externalizing symptoms when parents use noncontrolling parenting strategies.[14] In contrast, maternal positive discipline has been shown to predict fewer externalizing symptoms among temperamentally difficult children but not among easy children.[17] Recent evidence from molecular genetic research extends these child moderated models to illustrate how specific allelic polymorphisms (eg, DRD4, DRD2, HTR2A) condition the effects of parenting to influence child outcomes.[18-21]

Despite increasing evidence for the interplay between child and parenting characteristics, studies in this area have inferred genetic influences via the inclusion of child temperament or have focused on the interaction between parenting and a single polymorphism. Both design strategies explore only minimal genetic influences. In temperament models, genetic influences are inferred rather than measured, even though temperament exhibits low stability during infancy,[22] associations between temperament and behavior problems are highly correlated,[11] and associations likely arise due to a combination of environmental and genetic influences.[23] In molecular genetic models that examine Gene × Environment (G×E) interaction, genetic variation is typically measured via a single gene, and therefore leave open the likely possibility that many genetic influences remain unmeasured. In the present study, we extend prior work by examining the expressed effect of the whole genome using a quantitative behavioral genetic approach. In this approach, the full set of genetically influenced characteristics passed from biological parent to child can be examined by comparing similarities between birth parents and their reared apart child. As such, the extent to which effects of the whole genome interact with a discrete parenting behavior can be examined to further the understanding of processes whereby risk for psychopathology emerges during early childhood.

A prospective adoption design, where infants are adopted at birth and placed with nonrelative adoptive parents, allows the effects of parenting, genetic influences, and their interaction to be disentangled. In this design, in the absence of selective placement, similarities between adoptive parents and the adopted child are assumed to reflect environmental effects, and similarities between birth parents and the adopted child are assumed to reflect influences of the whole genome. Because distinct externalizing and internalizing factors often cannot be distinguished during early toddlerhood[24-25] but aggregate risk for psychopathology can be reliability measured,[26] the outcome under investigation in the present study was the toddler's total problems as predicted by the biological parents' psychopathology.

To date, only 1 other prospective full adoption study examining genetic and environmental effects on toddler behavior exists: The Colorado Adoption Project (CAP).[27] The CAP has yielded evidence of G×E interaction on psychopathology during later childhood[28-29] but not during infancy. In the present study, we built upon the methods of the CAP by measuring structured parenting via an observational assessment during toddlerhood, and by including multiple measures of birth parent psychopathology to increase the reliability of measurement of genetic risk. This theory-guided approach yields increased measurement precision for risk for psychopathology, allowing for the ability to detect G×E interaction as early as 9 months of age.[30]

In the present study, we tested two hypotheses: (a) that the main effect of structured parenting on toddler behavior problems would be apparent at 18 months of age, and (b) that the effects of structured parenting would vary depending on the child's genetic risk, such that children at high genetic risk as compared to children at low risk would differentially benefit (G×E interaction). Given associations between temperament and child behavior problems[11], we included indices of temperament risk at 9 months of age to control for the effects of prior behavioral risk and examine G×E interaction effects as they unfolded during toddlerhood.

Method

Participants

The sample consisted of linked adopted children, adoptive parents, and birth parents participating in the Early Growth and Development Study. The participants were recruited via adoption agencies in the Northwest, Mid-Atlantic, and Southwest regions of the United States. Infants were adopted by nonrelatives domestically within 3 months postpartum. The analytical sample included 290 linked triads (child, adoptive parent, and birth mother [BM]) who had data on all measures used in the present analyses. In addition, birth father (BF) data were included in a second set of analyses (n = 95). There were no differences in the variables used in this report for BMs with a participating BF and those without a participating BF. The children were 9 months old at the first assessment (Time 1 [T1]; M = 9.20, SD = .55), and 18 months old at the second assessment (Time 2 [T2]; M = 18.00 months, SD = 1.30 months). Forty-three percent of the children were female. The mean age of the child at the adoption placement was 3 days (SD = 5 days). The adoptive families were typically college educated and middle class, and the adoptive mother (AM) and adoptive father (AF) mean ages were 37 and 38, respectively. On average, the birth parents had high school or trade school education and household incomes under $25,000. The BM and BF mean ages were 24 and 25, respectively.

Measures

Birth parents were assessed in-person between 3- and 6-months postpartum (T1); adoptive families were assessed in-person at child age 9 (T1) and 18 (T2) months. Adoptive and birth parent participants completed questionnaires at all assessments. In addition, AMs were observed in a clean-up task with their child at T2. Participants were paid for their time. Additional details on the Early Growth and Development Study procedures, sample, and assessment methods can be found elsewhere.[31-32] The project was approved by the institutional review boards at George Washington University, the Oregon Social Learning Center, and the University of California, Davis, and the participants signed informed consent forms. For the present analyses, we selected indicators that minimized method overlap between the independent and dependent variables (eg, birth parent report, adoptive parent report, observed parenting).

Genetic Risk—Four measures of BM and BF psychopathology were collected via self-report at T1. Means and standard deviations for composite scores are presented in Table 1.

Alcohol, tobacco, marijuana, and other drug (ATOD) use: ATOD use was measured using a modified version of the Composite International Diagnostic Interview Short-Form Alcohol and Drug Dependence scales.[33] The modifications included a set of tobacco dependence questions and a lifetime use response frame. The indicators of lifetime problem use of alcohol, tobacco, marijuana, and other drugs were created, standardized, and combined to form a composite score (BM α = .72, BF α = .71).

Antisociality: Antisociality was measured using the 38-item Elliot Social Behavior Questionnaire.[34] Birth parents self-reported on their engagement in antisocial behaviors. Items were summed to create an antisocial index (BM α = .88, BF α = .91), and scores were log-transformed to reduce skewness.

Depression: Depression was measured using the 21-item Beck Depression Inventory[35] and was calculated as the sum of 20 of the items (BM α = .92, BF α = .89); the suicidal ideation item was not administered to minimize situations where clinical follow-up would be necessitated.

Anxiety: Anxiety was measured using the 21-item Beck Anxiety Inventory[36] and was calculated as the sum of the items (BM α = .90, BF α =.88).

Associations among the birth parent indicators: Correlations among the psychopathology indicators ranged from .27–.69 (BM) and .16–.60 (BF). The indicators were standardized within parent, and a composite score was formed by taking the mean of the indicators. The BM and BF psychopathology constructs were modestly correlated (r = .27). Higher scores indicated greater psychopathology.

Structured Parenting—At T2, mother–child dyads participated in a structured 3-minute clean-up task. The interviewer asked the AM to have her child clean up a number of multi-piece toys (eg, a shape sorter, a hide-inside soft box, and a set of stacking rings) and put each toy set in its separate container. The AM was instructed to make sure that only the child cleaned up and to remind him/her as necessary. The task was coded from DVD using the Parent–Child Free Play and Compliance Task Coding Manual (Pears & Ayers, unpublished coding manual, 2005), a 4-digit, real-time microsocial system that indicates the initiator, the initiator's behavior (2-digit), and the recipient. When an observation code changes, a new 4-digit code is entered.

Four parenting verbal behavior codes occurred with adequate frequency in the present study (positive reinforcement, on-task talk, off-task talk, and parental requests). We analyzed the *parental request* code due to its theoretical relevance to toddler behavior problems and observed associations with child outcomes in prior studies, and computed the duration of time that the mother spent giving requests to the child (ie, "structured parenting"). Structured parenting included all types of statements in which a behavior change was suggested, including questions (eg, "Where does this ring go?"), statements (eg, "Let's put the duck in this box."), and directives (eg, "Put all of the cups in here."). A defining feature was that there was an explicit or implicit behavior change or a specific action desired of the child. Fifteen percent of the tapes were coded by 2 independent coders; the average intercoder agreement on the behavior content code was .88% (overall κ = .71).

Toddler Behavior Problems—At T2, AMs completed the Child Behavior Checklist[37], which consists of 99 behaviors rated on a 3-point scale with values of 0 (*not true*), 1 (*sometimes true*), and 2 (*very true*). The total problems T-score, which sums all 99 items, was used in the present analyses (α = .90), thereby indicating general risk for psychopathology in a parallel manner to the broadband indicator of birth parent psychopathology.

Covariates

Adoption openness: To control for similarities between birth and adoptive families that might result from contact between parties, T1 level of openness in the adoption was measured using a composite of BM, BF, AM, and AF perceived adoption openness rated on a 7-point scale: 1 (*very closed*) to 7 (*very open*). Interrater agreement was high (*r* range = .66–.81).[38]

BM prenatal ATOD use: Because prenatal ATOD use can confound estimates of genetic influences, the BMs reported their prenatal use of 10 substance classes (ie, tobacco, alcohol, sedatives, tranquilizers, amphetamines, painkillers, inhalants, cocaine, heroin, and hallucinogens) at T1, using a pregnancy history calendar.[39] All 10 items were dichotomized, Cronbach's α (KR-20) = .67. The sum of dichotomous indicators was positively skewed and collapsed into a 5-point scale: 0 (*prenatal use of no substances*) to 4 (*prenatal use of four or more substances*).

Infant temperament risk: To account for the contributions of prior temperamental risk on toddler behavior problems, AMs and AFs completed the Infant Behavior Questionnaire[40] at T1. The Distress to Limitations (20 items) and Fear (16 items) subscales were used because of their documented associations with later externalizing and internalizing symptoms, respectively[11-13]: 1 (*never*) to 7 (*always*). Inter-item reliability for the Distress to Limitations subscale was .73 for AMs and AFs; inter-item reliability for the Fear subscale was .73 for AMs and .71 for AFs. The correlations between AM and AF ratings were high (range = .52 to .59); therefore, a composite score (ie, mean of the 2 parents' ratings) was computed to measure temperament risk.

Data Analysis

The analyses were conducted to examine genetic (birth parent psychopathology), parenting, and G×E interaction effects on toddler behavior problems. Ordinary least squares regression analyses were conducted in SPSS using listwise deletion in the prediction models. A hierarchical approach was employed: Step 1 included the control variables (openness in adoption, prenatal ATOD use, infant distress to limitations, and infant fearfulness), Step 2 added birth parent psychopathology and structured parenting, and Step 3 added the G×E interaction. Two separate regression models were examined. First, BM psychopathology was used as the indicator of genetic risk (listwise N = 290). Second, BF psychopathology was used (listwise n = 95) as a means of replicating the pattern of findings obtained with the BM data. Following procedures recommended by Aiken and West[41] for a conservative estimation of interaction effects, when an individual interaction term was significant, we examined the direction of effects by splitting the sample at 1 standard deviation above and below the mean on the birth parent variable and plotting the effects of structured parenting on toddler problems within the resulting subsamples.

Results

Descriptive Statistics

Correlational analyses indicated that T2 toddler problems were positively associated with T1 temperament risk and openness, and were negatively associated with T2 structured parenting. In addition, BM and BF psychopathology scores were associated with one another and with BM prenatal ATOD use. Neither BM psychopathology, BF psychopathology, or infant temperament risk was significantly associated with structured parenting, suggesting the negligible role of evocative gene–environment correlation as an explanatory mechanism for the association between parenting and toddler problems. That is, genetically-influenced

characteristics of the child did not appear to systematically relate to the level of structured parenting from AMs.

Regression Model Predicting Toddler Behavior Problems From BM Psychopathology

The hierarchical regression model predicting toddler behavior problems from BM psychopathology, structured parenting, and their interaction is presented in Table 2. Step 1 was significant, $F(4, 285) = 13.26$, $p < .001$, and indicated an effect of higher distress to limitations, $\beta = .35$, $p < .001$, and higher levels of openness, $\beta = .15$, $p < .01$, on toddler problems. Step 2 resulted in a trend for a significant increase in the percentage of variance explained, $F(6, 283) = 9.94$, $p < .001$, $\Delta R^2 = .017$, $p < .10$. The 2 covariates from Step 1 retained their contributions and there was a significant inverse effect of structured parenting, $\beta = -.12$, $p < .05$ on toddler problems. Step 3 included the interaction term, which was also statistically significant, $\beta = -.20$, $p < .001$. Moreover, adding the interaction term to the model reduced the main effect of structured parenting to a trend, $F(7, 282) = 10.83$, $p < .001$, $\Delta R^2 = .038$, $p < .001$.

As is shown in Figure 1, the decomposition of the interaction indicated that the effect of structured parenting varied based on genetic risk. Two distinct pathways to risk for psychopathology were identified. In the first, structured parenting buffered the negative effects of genetic risk on toddler behavior problems: increased structured parenting reduced child problems when the BM had higher levels of psychopathology. In the second pathway, structured parenting enhanced risk for behavior problems for children at low genetic risk. Examination of the interaction effects with the mean split conferred a similar pattern of effects.

Regression Model Predicting Behavior Problems From BF Psychopathology

A similar regression model was estimated using BF psychopathology as the genetic indicator. This model was employed to potentially validate the BM findings using an alternate source of genetic risk. As is shown in Table 3, Step 1 indicated a significant effect of infant distress to limitations, $\beta = .42$, $p < .001$, and openness, $\beta = .21$, $p < .05$, on toddler problems, $F(4, 90) = 8.63$, $p < .001$. Step 2 remained significant, $F(6, 88) = 5.73$, $p < .001$, although neither BF psychopathology or structured parenting reached statistical significance. The interaction term was added in Step 3, $F(7, 87) = 7.74$, $p < .001$, and was significant, $\Delta R^2 = .035$, $p < .05$.

The BF interaction effect is shown in Figure 2 for the 2 subgroups: 1 standard deviation above/below the mean on BF psychopathology. Similar to the BM interaction effect, two pathways of risk were identified. Low levels of structured parenting were associated with behavior problems for toddlers at high genetic risk, whereas high levels of structured parenting were associated with behavior problems for toddlers at low genetic risk. The pattern of effects was similar when decomposed via a mean split.

Discussion

The central finding of this study is that the effects of structured parenting on toddler behavior problems were conditioned by birth parent psychopathology, suggesting that different types of parenting influence risk for psychopathology for toddlers at high versus low genetic risk. When BM psychopathology was high, structured parenting helped to offset toddler behavior problems. The same buffering effect of structured parenting was found when BF psychopathology was high. Conversely, when BM or BF psychopathology was low, structured parenting *increased* toddler problems.

This interaction suggests the possibility of two distinct pathways to early risk for psychopathology. In the first pathway, structured parenting may be beneficial for children at high genetic risk. This is consistent with findings from preventive intervention studies

demonstrating greater impact with higher risk children.[42-44] However, a second pathway indicated that structured parenting can be a risk factor for children at low genetic risk. For these children, high levels of structured parenting were associated with increased toddler problems. Providing high levels of structured parenting to children at low risk might have a detrimental effect because such children do not need extensive structuring, which might impede or intrude upon their normative development. As such, toddlers' inheritance may not be high or low "risk," but rather, a suitability for a highly structured or less structured parenting environment.[45] The present study extends prior research by illuminating genetic influences as a specific mechanism that might account for the differential effectiveness of programs designed to improve parenting practices.

The G×E interaction finding (using BM data) from this study is strengthened by the validation of the interaction pattern using BF psychopathology as the index of genetic influences. Further, a discrete aspect of parenting was observationally coded, thereby eliminating method and informant overlap between the environment, outcome, and genetic measures and affording the opportunity to examine a specific parenting behavior that could be targeted via intervention. Additionally, the inclusion of T1 temperament risk in the analytical models facilitated two purposes. First, associations between temperament risk and later behavioral problems could be examined. Replicating studies of biological families[11], the present study found significant associations between temperamental risk and later behavior problems, although both traits were rated by parents, which may have artificially inflated associations. Second, the inclusion of T1 temperamental risk allowed for the specification of the timing of the G×E interaction to unfold in the period between 9 and 18 months—a developmental period when children become increasing mobile and verbal, and structured parenting therefore a more critical aspect of parenting. Lastly, the inclusion of prenatal ATOD use in the analyses reduced the likelihood that the associations found resulted from prenatal drug exposure, a common confound in studies of risk for psychopathology.[46]

Partial support was also provided for the hypothesized main effect of structured parenting, controlling for temperament risk. Structured parenting was inversely related to toddler problems in the BM model, and its effect was reduced to a trend in the presence of the interaction term, suggesting genetic moderation. The main effect of parenting on toddler problems adds to the body of research on such linkages[7,9] by suggesting associations in families where the parent and child are not genetically related. As such, the role of shared genes as a mediating mechanism underlying associations between parent and child can be ruled out.

Of note, although genetic risk for psychopathology in this sample was high (eg, 20% of BMs and 17% of BFs had scores in the moderate-to-severe range on the Beck Anxiety Inventory (above 15), and over half of the birth parents reported use of at least 1 illicit drug class), the toddlers generally exhibited normative level of problems (CBCL T-score $M = 46$), suggesting the relatively strong impact of the environment during early childhood. Adoptive parents varied in the extent to which they used structured parenting behaviors. On average, parents engaged in structured parenting about one-third of the interaction time, although some parents never engaged in such behaviors and others did so for the entire interaction period. The high impact of structured parenting found in the present study, combined with the large variability in this measure, suggest the potential for developing interventions that aim to increase or decrease levels of structured parenting at an individual level (depending upon the child's genetic risk).

This study had several limitations. First, although this is the largest existing BF sample in a full adoption design and it provided converging support for the G×E interaction, our sample size was reduced for the BF analyses. Second, our measure of child behavior problems—the CBCL—was not as sensitive to identifying early risk for psychopathology as other measures might be (e.g., the ITSEA).[47] As such, associations between the CBCL with structured

parenting and BP psychopathology scores may have been attenuated. In addition, because of the lack of delineation between externalizing and internalizing constructs at 18 months of age, we examined total behavior problems. Studies later in childhood, when the etiological precursors and current correlates of externalizing and internalizing disorders become more discrete, could provide important specificity about distinct pathways. In addition, longitudinal studies would allow for the assessment of the extent to which structured parenting has long-term effects on risk for psychopathology. Finally, the observational assessment was relatively short, and it is unclear how the interaction effects would generalize to other compliance situations and less structured contexts.

Notwithstanding these limitations, the results from the present study underscore the importance of examining the effects of the whole genome via transmission from birth parent to child when considering the effects of parenting. In particular, the findings highlight the role of genetic contributions to toddler's suitability to specific parenting environments, with children at genetic risk for psychopathology benefiting from more structured environments and children at low risk for psychopathology benefitting from less structured environments. As such, consideration of the individual, inherited risks a toddler presents with might be warranted when implementing interventions aimed at preventing risk for psychopathology during early childhood.

Acknowledgments

Dr. Leve is at the Oregon Social Learning Center, Eugene, OR; Dr. Harold is at the University of Otago, Dunedin, New Zealand; Dr. Ge is at the University of Minnesota; Dr. Neiderhiser is at The Pennsylvania State University; Dr. Shaw is at the University of Pittsburgh; Dr. Scaramella is at the University of New Orleans; Dr. Reiss is at the Yale Child Study Center.

Support for this project and the writing of this manuscript was provided by the following grants: R01 HD042608 and P30 DA023920. Special gratitude is given to Rand Conger and John Reid for their contributions to this study.

References

1. Caspi A, Moffitt TE, Newman DL, Silva PA. Behavioral observations at age 3 years predict adult psychiatric disorders: longitudinal evidence from a birth cohort. Arch Gen Psychiatry 1996;53:1033–1039. [PubMed: 8911226]
2. Shaw DS, Owens EB. Infant and toddler pathways leading to early externalizing disorders. J Am Acad Child Adolesc Psychiatry 2001;40:36–43. [PubMed: 11195559]
3. Denham SA, Workman E, Cole PM, Weissbrod C, Kendziora KT, Zahn-Waxler C. Prediction of externalizing behavior problems from early to middle childhood: the role of parental socialization and emotion expression. Dev Psychopathol 2000;12:23–45. [PubMed: 10774594]
4. Gardner FEM, Sonuga-Barke EJS, Sayal K. Parents anticipating misbehaviour: An observational study of strategies parents use to prevent conflict with behaviour problem children. J Child Psychol Psyc 1999;40:1185–1196.
5. Holden GW. Avoiding conflict: mothers as tacticians in the supermarket. Child Dev 1983;54:233–240.
6. Holden GW, West MJ. Proximate regulation by mothers: a demonstration of how differing styles affect young children's behavior. Child Dev 1989;60:64–69. [PubMed: 2702875]
7. Lecuyer E, Houck GM. Maternal limit-setting in toddlerhood: Socialization strategies for the development of self-regulation. Infant Ment Health J 2006;27:344–370.
8. Dishion TJ, Shaw DS, Connell A, Wilson MN, Gardner F, Weaver C. The Family Check Up with high-risk families with toddlers: outcomes on positive parenting and early problem behavior. Child Dev 2008;79:1395–1414. [PubMed: 18826532]
9. Gardner FEM, Dishion TJ, Shaw DS, Burton J, Supplee L. Randomized prevention trial for early conduct problems: effects on proactive parenting and links to toddler disruptive behavior. J Fam Psychol 2007;21:398–406. [PubMed: 17874925]

10. Caspi A, Harrington H, Milne B, Amell JW, Reremoana F, Moffitt TE. Children's behavioral styles at age 3 are linked to their adult personality traits at age 26. J Pers 2003;71:495–513. [PubMed: 12901429]
11. Lemery KS, Essex MJ, Smider NA. Revealing the relations between temperament and behavior problem symptoms by eliminating measurement confounding: expert ratings and factor analysis. Child Dev 2002;73:867–882. [PubMed: 12038557]
12. Feng X, Shaw DS, Silk JS. Developmental trajectories of anxiety symptoms among boys across early and middle childhood. J Abnorm Psychol 2008;117:32–47. [PubMed: 18266484]
13. Leve LD, Kim HK, Pears KC. Childhood temperament and family environment as predictors of internalizing and externalizing trajectories from ages 5 to 17. J Abnorm Psychol 2005;35:505–520.
14. Bates JE, Pettit GS, Dodge KA, Ridge B. Interaction of temperamental resistance to control and restrictive parenting in the development of externalizing behavior. Dev Psychol 1998;34:982–995. [PubMed: 9779744]
15. Morris AS, Silk JS, Steinberg L, Sessa FM, Avenevoli S, Essex MJ. Temperamental vulnerability and negative parenting as interacting of child adjustment. J Mar Fam 2002;64:461–471.
16. Belsky J, Hsieh KH, Crnic K. Mothering, fathering, and infant negativity as antecedents of boys' externalizing problems and inhibition at age 3 years: differential susceptibility to rearing experience. Dev Psychopathol 1998;10:301–319. [PubMed: 9635226]
17. van Zeijl J, Mesman J, Stolk MN, Alink, et al. Differential susceptibility to discipline: the moderating effect of child temperament on the association between maternal discipline and early childhood externalizing problems. J Fam Psychol 2007;21:626–636. [PubMed: 18179334]
18. Bakermans-Kranenburg MJ, van Ijzendoorn MH. Gene–environment interaction of the dopamine D4 receptor (DRD4) and observed maternal insensitivity predicting externalizing behavior in preschoolers. Dev Psychobiol 2006;48:406–409. [PubMed: 16770765]
19. Jokela M, Keltikangas-Järvinen L, Kivimäki M, et al. Serotonin receptor 2A gene and the influence of childhood maternal nurturance on adulthood depressive symptoms. Arch Gen Psychiatry 2007;64:356–360. [PubMed: 17339524]
20. Mills-Koonce WR, Propper CB, Gariepy JL, Blair C, Garrett-Peters P, Cox MJ. Bidirectional genetic and environmental influences on mother and child behavior: The family system as the unit of analysis. Dev Psychopathol 2007;10:1073–1087. [PubMed: 17931435]
21. Sheese BE, Voelker PM, Rothbart MK, Posner MI. Parenting quality interacts with genetic variation in dopamine receptor D4 to influence temperament in early childhood. Dev Psychopathol 2007;19:1039–1046. [PubMed: 17931433]
22. Lemery KS, Goldsmith HH, Klinnert MD, Mrazek DA. Developmental models of infant and childhood temperament. Dev Psychol 1999;35:189–204. [PubMed: 9923474]
23. Roisman GI, Fraley RC. The limits of genetic influence: A behavior-genetic analysis of infant–caregiver relationship quality and temperament. Child Dev 2006;77:1656–1667. [PubMed: 17107452]
24. Gjone H, Stevenson J. The association between internalizing and externalizing behavior in childhood and early adolescence: genetic or environmental common influences? J Abnorm Child Psychol 1997;25:277–286. [PubMed: 9304444]
25. van den Oord EJCG, Boomsma DI, Verhulst FC. A study of genetic and environmental effects on the co-occurrence of problem behaviors in three-year-old twins. J Abnorm Psychol 2000;109:360–372. [PubMed: 11016106]
26. Essex MJ, Kraemer HC, Armstrong JM, et al. Exploring risk factors for the emergence of children's mental health problems. Arch Gen Psychiatry 2006;63:1246–1256. [PubMed: 17088505]
27. Plomin, R.; DeFries, JC.; Fulker, DW., editors. Nature and Nurture During Infancy and Early Childhood. Cambridge University Press; New York: 1988.
28. Hershberger, SL. Genotype–environment interaction and correlation. In: DeFries, JC.; Plomin, R.; Fulker, DW., editors. Nature and Nurture During Middle Childhood. Blackwell; Malden, MA: 1994. p. 281-294.
29. O'Connor TG, Caspi A, DeFries JC, Plomin R. Genotype–environment interaction in children's adjustment to parental separation. J Child Psychol Psychiatry 2003;44:849–856. [PubMed: 12959493]

30. Leve LD, Kerr D, Shaw D, et al. Infant pathways to externalizing behavior: evidence of Genotype × Environment interaction. Child Dev. In press
31. Leve LD, Neiderhiser JM, Scaramella LV, Reiss D. The Early Growth and Development Study: using the prospective adoption design to examine genotype–environment interplay. Acta Psychol Sin 2008;40:1106–1115.
32. Leve LD, Neiderhiser JM, Ge X, et al. The Early Growth and Development Study: a prospective adoption design. Twin Res Hum Genetics 2007;10:84–95. [PubMed: 17539368]
33. Kessler R, Andrews G, Mroczek D, Ustun B, Witchen H. The World Health Organization Composite International Diagnostic Interview Short-Form (CIDI-SF). Int J Methods Psychiatr Res 1998;7:171–184.
34. Elliot, DS.; Huizinga, D.; Ageton, SS. Explaining Delinquency and Drug Use. Sage; Beverly Hills, CA: 1985.
35. Beck, A.; Steer, R. Beck Depression Inventory Manual. Psychological Corporation; San Antonio, TX: 1993.
36. Beck, A.; Steer, R. Beck Anxiety Inventory Manual. Psychological Corporation; San Antonio, TX: 1993.
37. Achenbach, TM.; Rescorla, LA. Manual for the ASEBA Preschool Forms & Profiles. University of Vermont, Research Center for Children, Youth, & Families; Burlington, VT: 2000.
38. Ge X, Natsuaki MN, Martin D, et al. Bridging the divide: openness in adoption and post-adoption psychosocial adjustment among birth and adoptive parents. J Fam Psychol 2008;22:529–540. [PubMed: 18729667]
39. Caspi A, Moffit TE, Thornton A, et al. The life history calendar: a research and clinical assessment method for collecting retrospective event-history data. Int J Methods Psychiatr Res 1996;6:101–114.
40. Rothbart MK. Measurement of temperament in infancy. Child Dev 1981;52:569–578.
41. Aiken, LS.; West, SG. Multiple Regression: Testing and Interpreting Interactions. Sage; Newbury Park, CA: 1991.
42. Shaw DS, Dishion TJ, Supplee LH, Gardner F, Arnds K. A family-centered approach to the prevention of early-onset antisocial behavior: two-year effects of the family check-up in early childhood. J Consult Clinic Psychol 2006;74:1–9.
43. Ialongo NS, Werthamer L, Kellam SG, Brown CH, Wang S, Lin Y. Proximal impact of two first-grade preventive interventions on the early risk behaviors for later substance abuse, depression, and antisocial behavior. Am J Community Psychol 1999;27:599–641. [PubMed: 10676542]
44. Stoolmiller M, Eddy JM, Reid JB. Detecting and describing preventive intervention effects in a universal school-based randomized trial targeting delinquent and violent behavior. J Consult Clin Psychol 2000;68:296–306. [PubMed: 10780130]
45. Kendler KS, Eaves LJ. Models for the joint effect of genotype and environment on liability to psychiatric illness. Am J Psychiatry 1986;143:279–289. [PubMed: 3953861]
46. Rice F, Harold GT, Boivin J, Hay D, van den Bree MMB, Thapar A. Disentangling prenatal and inherited influences in humans with an experimental design. Proc of the Natl Acad of Sci 2009;1061:2464–2467.
47. Carter AS, Briggs-Gowan MJ, Jones SM, Little TD. The Infant-Toddler Social and Emotional Assessment (ITSEA): Factor Structure, Reliability, and Validity. J of Abnorm Child Psychol 2003;31:495–514. [PubMed: 14561058]

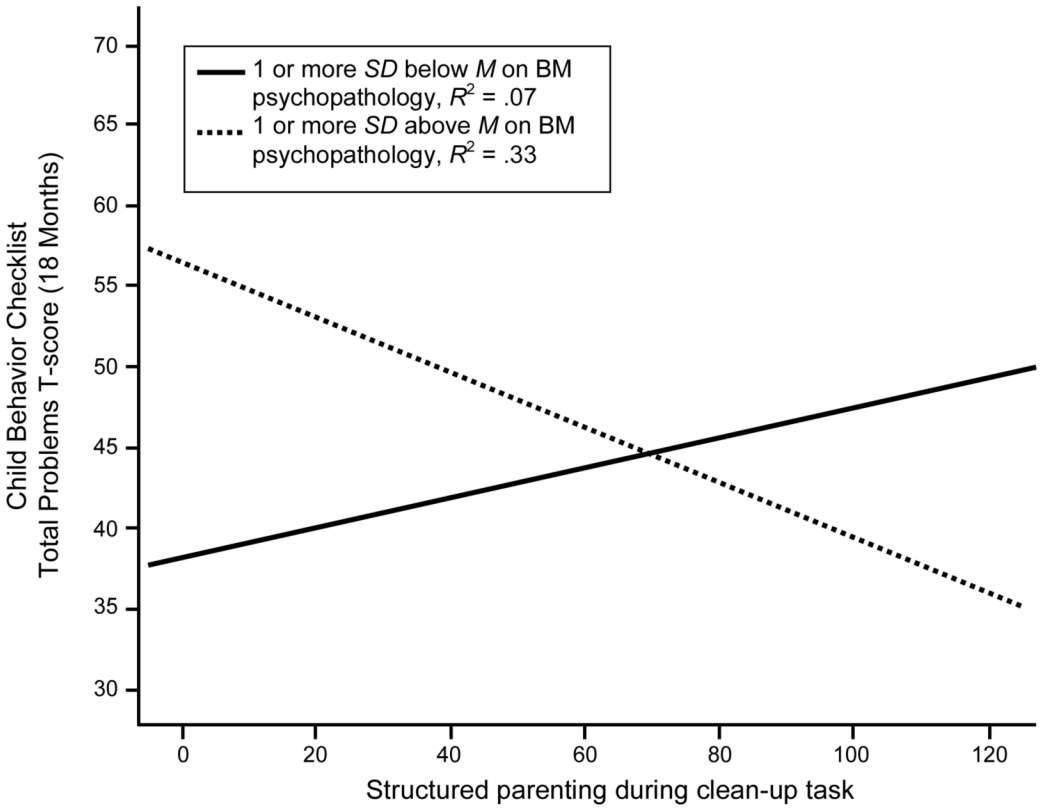

Figure 1.
Interaction between birth mother (BM) psychopathology and structured parenting on toddler behavior problems. Note: BM, birth mother.

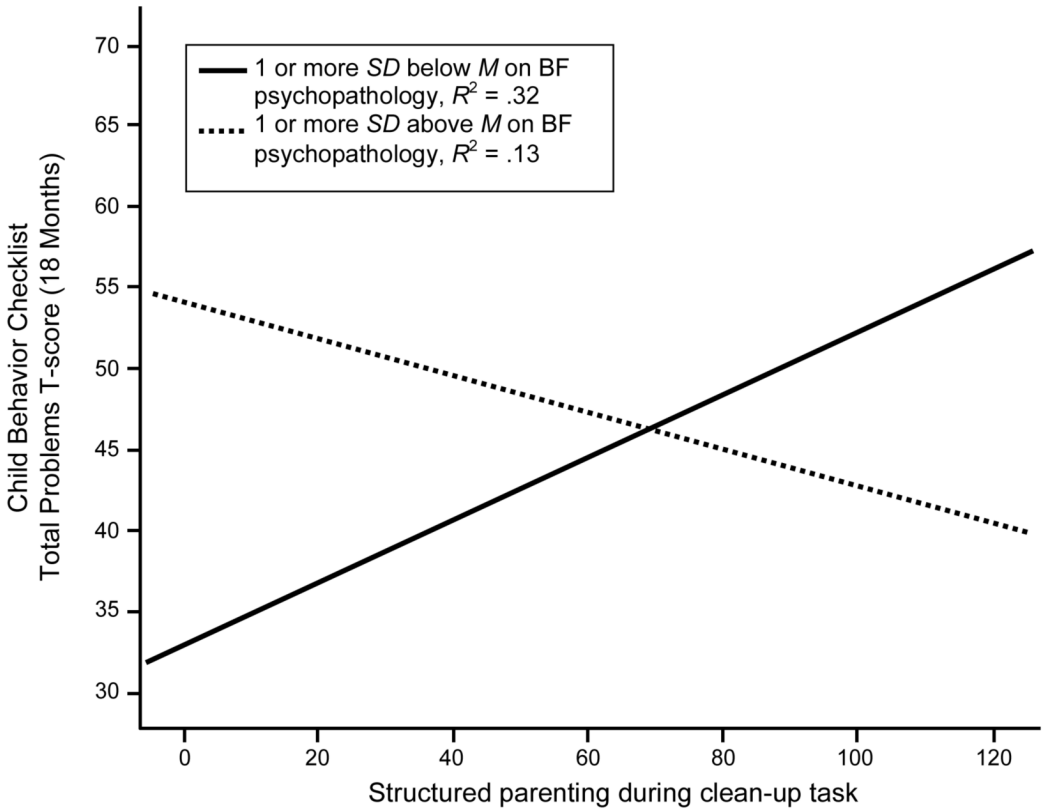

Figure 2.
Interaction between birth father (BF) psychopathology and structured parenting on toddler behavior problems. Note: BF, birth father.

Table 1

Means, Standard Deviations, and Correlations Among Variables Used in Regression Analyses

Variable	1	2	3	4	5	6	7	8
1. Openness (T1)								
2. Prenatal ATOD use (T1)	.05							
3. Infant distress to limitations (T1)	.03	.03						
4. Infant fearfulness (T1)	−.10^{+}	−.08	.38***					
5. BM psychopathology (T1)	−.02	.46***	−.01	−.06				
6. BF psychopathology (T1)	.04	.30**	−.03	−.12	.27**			
7. Structured parenting (T2)	.08	.00	−.04	−.01	−.01	−.08		
8. Toddler behavior problems (T2)	.14*	.07	.34***	.14*	.05	−.09	−.12*	
M	4.52	1.01	.00	−.01	.00	−.02	64.20	45.77
SD	1.16	1.17	.90	0.91	.76	.74	22.12	7.42

Abbreviations: ATOD, alcohol, tobacco, and other drugs; BF, birth father; BM, birth mother; T1, Time 1; T2, Time 2.

$^{+}p < .10$

$^{*}p < .05$

$^{**}p < .01$

$^{***}p < .001$.

Table 2

Hierarchical Regression Model Predicting T2 Toddler Behavior Problems Using BM Psychopathology as the Index of Genetic Risk

Variable	Step 1 β	Step 2 β	Step 3 β
Adoption openness (T1)	.15**	.16**	.15**
BM prenatal ATOD use (T1)	.04	.01	.02
Infant distress to limitations (T1)	.35***	.35***	.33***
Infant fearfulness (T1)	.06	.06	.06
BM psychopathology (T1)		.07	.07
Structured parenting (T2)		−.12*	−.09+
G×E			−.20***

Note: Final step model, $F(7, 282) = 10.83^{***}$, $R^2 = .21$; ΔR^2 in Step 2 = .017, $p < .06$; ΔR^2 in Step 3 = .038, $p < .001$.

Abbreviations: BM, birth mother; G×E, Gene × Environment interaction; T1, Time 1; T2, Time 2.

+ $p < .10$

* $p < .05$

*** $p < .001$.

Table 3

Hierarchical Regression Model Predicting T2 Toddler Behavior Problems Using BF Psychopathology as the Index of Genetic Risk

Variable	Step 1 β	Step 2 β	Step 3 β
Adoption openness (T1)	.21*	.20*	.20*
BM prenatal ATOD use (T1)	−.03	−.03	−.03
Infant distress to limitations (T1)	.42***	.43***	.38**
Infant fearfulness (T1)	.07	.08	.07
BF psychopathology (T1)		−.01	−.02
Structured parenting (T2)		.06	.08
G×E			−.19*

Note: Final step model, $F(7, 87) = 5.74^{***}$, $R^2 = .32$; ΔR^2 in Step 2 = .004, *ns*; ΔR^2 in Step 3 = .035, $p < .05$.

Abbreviations: BF, birth father; BM, birth mother; G×E, Gene × Environment interaction; T1, Time 1; T2, Time 2.

* $p < .05$

** $p < .01$

*** $p < .001$.