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Predicting Caregiver-Reported Behavior Problems in Cocaine-Exposed Children at 3 Years

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Abstract

Predictors of caregiver-reported behavior problems for 3-year-olds with prenatal cocaine exposure (PCE) and matched controls were examined using structural equation modeling. We tested whether PCE had a direct effect on child behavior problems in a model that included other prenatal drug exposure, child sex, caregiver depression, and the quality of the child's home environment. The sample (N = 256) was drawn from a longitudinal, prospective study of children of (predominantly crack) cocaine-using women and controls matched on race, socioeconomic status, parity, and pregnancy risk. Child Behavior Problems was modeled as a latent variable composed of the 48-item Conners' Parent Report Scale Conduct Problem and Impulsive-Hyperactive scales and the Eyberg Child Behavior Inventory Intensity scale. Caregiver depression was the only significant predictor of Child Behavior Problems. Mean levels of caregiver self-reported depression and reported child behavior problems did not differ between groups. Mean depression scores were well above the recommended clinical cutoff while mean child behavior problems scores were within normal limits. The model explained 21% of the variance in caregiver-reported child behavior problems in our sample of rural African American, low SES youngsters. Non-maternal caregivers of cocaine-exposed children had significantly lower mean depression scores and mean child behavior problems ratings for 2 of 3 scales used in the study compared to biological mothers of children with PCE and controls. For all groups, much larger proportions of children were rated as having clinically significant behavior problems than would be expected based on the prevalence of behavior problems in the general population.

Since the coining of the term "crack baby" in the 1980s, dire predictions have been made about the developmental outcomes of children with prenatal cocaine exposure (PCE). These premature forecasts were based, in part, on research conducted using animal models in which considerable deleterious effects of PCE on offspring were found as well as in case reports and clinical studies of polydrug users. Early controlled studies of neonates and young infants with PCE did indicate difficulties with state and behavioral regulation including problems with alertness, orientation, irritability, and arousal-modulated attention which raised the concern that these children could be at risk for externalizing behavior problems in the future.¹ While well-designed prospective studies in humans during the past decade have yielded more equivocal results, most studies have been consistent with the conclusion that PCE exerts clinically subtle but statistically significant effects that could result in considerable costs to society.²

The literature on child behavior problems in children with PCE, as with other developmental outcomes, is plagued by conflicting findings. Not one of seven published studies using parent-report measures of behavior problems have found significant effects of PCE.³⁻⁵ In contrast, four of the five studies using teacher-report measures have reported significant findings.⁶⁻⁹ However, all were based on the same cohort.

One theme that appears to be emerging from the literature is the important influence of postnatal environmental factors, particularly maternal psychological functioning, on reported behavior problems of children with PCE. Maternal depression was found to be significantly related to externalizing problem scores of 2-year-olds with PCE on the Child Behavior Checklist (CBCL¹⁰), and maternal stress and lack of social support were associated with higher scores on both the internalizing and externalizing scales.¹¹ Custody changes, the child's report of exposure to violence, and current drug use in the home were related to Conners' Teacher Report Form (TRF¹⁰) scores for 6-year-olds with PCE.⁷ Accornero et al.⁴ reported that recent maternal drug use and maternal psychological distress were associated with CBCL externalizing and internalizing problems at age 5. Bennett and colleagues⁵ found that maternal self-report of harsh discipline, depressive symptoms, and a composite variable of environmental risk significantly predicted CBCL externalizing problems while maternal depression predicted CBCL internalizing problems at age 4. Considering the high rate of comorbidity between depression and crack cocaine use found in community-based samples,^{13, 14} it is not surprising that studies of children with PCE have found an association between maternal depression and report of child behavior problems.

The purpose of the current study was to examine predictors of caregiver-reported behavior problems in a sample of 3-year-old children with PCE and matched controls using two measures not previously reported in the literature, the 48-item Conners' Parent Report Scale (CPRS-48)¹⁴ and the Eyberg Child Behavior Inventory (ECBI).¹⁵ Unlike omnibus measures like the CBCL, which are designed to assess child behavior functioning across a variety of domains, the CPRS-48 has specific scales assessing conduct problems and impulsive/hyperactive behaviors. Similarly, the ECBI was designed specifically to assess disruptive behaviors, particularly those associated with oppositional-defiant and conduct disorders. We hypothesized that: (1) PCE, maternal/caregiver depression, and the quality of the child's home environment would each have a direct effect on reported child behavior problems, (2) PCE would have an indirect effect on reported child behavior problems through its relationship with maternal/caregiver depression and the quality of the child's home environment, and (3) maternal/caregiver depression would have an indirect effect on reported child behavior problems through its relationships with the quality of the child's home environment.

A secondary aim of the current study was to compare biological mothers who retained custody of their children and non-maternal caregivers of children with PCE in terms of depressive symptoms and ratings of child behavior. To our knowledge, all previous studies based on parent report measures of behavior problems in children with PCE have used biological mothers as raters. Due to the high rate of comorbidity between substance abuse and depression, we expected that the biological mothers of the children with PCE in our sample may experience significant levels of depression which would, in turn, influence their ratings of child behavior problems. Non-maternal caregivers, who presumably are drug-free, would be less likely to experience depression and could perhaps provide a more "objective" snapshot of the behavioral functioning of the children with PCE in their custody.

METHODS

Procedures

Approval for this prospective, longitudinal study was granted by our Institutional Review Board, and a federal Certificate of Confidentiality from the U.S. Department of Health and Human Services National Institute on Drug Abuse was obtained to protect the confidentiality of the data provided by participants. Amount and frequency of drug use was obtained during maternal interviews that covered the period of three months prior to the pregnancy and each trimester of the pregnancy. Using a procedure adapted from that of Day and colleagues,¹⁶ interviews were conducted by experienced female research staff at the end of each trimester or after birth, in the case of no prenatal care. Data analyses for this study are based on the children and their primary caregivers completing the study measures at the 3-year follow-up evaluation. When the children were 3 years old, experienced female research staff conducted extensive 1½-hour interviews in the home with the child's primary caregiver regarding the caregiver's current psychological functioning and the child's current behavior. Informed consent was obtained from the primary caregiver at the beginning of the visit. To avoid difficulties due to low literacy level of the caregivers, questionnaires were presented in an interview format with responses to Likert-scale items cued by a card showing the response choices. For their participation, caregivers were given age-appropriate toys for the child in the study.

Participants

Sample Recruitment and Retention—Detailed information regarding the recruitment and enrollment of participants in the longitudinal study has been published previously.¹⁷ Briefly, more than 2,500 potential participants were approached when they first entered the prenatal care system or, in the case of no prenatal care, when they presented to deliver. Experienced research staff carefully obtained informed consent from 85% of the mothers, who were then interviewed in depth. A priori exclusion criteria included several factors known to affect pregnancy or developmental outcome: major illnesses diagnosed prior to pregnancy, chronic use of prescription or over-the-counter drugs, any use of illicit drugs other than cocaine and marijuana, and <18 years old. Non-English speakers, who comprised less than 5% of the delivery population, were also excluded because the assessments were not validated for non-English speakers. Cocaine users were identified by private, structured interviews and/or urine specimens collected at two unanticipated times. Positive urine screens were confirmed by gas chromatography/mass spectroscopy.

The screening and selection process identified 154 (predominantly crack) cocaine using pregnant women. A comparison group of 154 non-using women was composed by matching each cocaine-using woman on four variables: race, socioeconomic status (SES), parity and location of prenatal care (as a measure of prenatal risk). The sample of 308 women was predominantly African American, in the lowest Hollingshead SES category,¹⁸ had more than one child, and the majority were not referred to a high-risk prenatal clinic for treatment. There were four fetal deaths and six subsequent deaths, resulting in a final cohort of 298 children of whom approximately 90% have been followed since birth.

Current Sample—Of the surviving 298 families originally enrolled, 256 (86%) participated in the 3-year assessment and completed all of the outcome measures. Reasons for families not participating at this study time were as follows: two had moved out of the area and an assessment could not be scheduled, five refused assessment at 3 years, 13 could not be located for follow up, and two dropped out of the study permanently. An additional 20 families were excluded from this report because data were missing for the child behavior measures. Chi-square and *t*-test comparisons between the 256 participants in the current

study and the 40 participants who were excluded or dropped out revealed no significant group differences in terms of race, SES, parity, exposure status (cocaine vs. not) or amount of cocaine exposure (all p 's > .05).

Of the 256 children included in this study, 123 were cocaine-exposed and 133 were not exposed to cocaine. Both groups were exposed to various amounts of tobacco, alcohol, and marijuana. As in the original cohort, participants in the current study did not differ on the four variables (race, SES, parity, and prenatal risk) used to match cocaine-using mothers with controls. Additionally, there were no significant differences in the mean age between the cocaine-exposed and non-exposed children at the time of assessment (3.09 ± 0.13 vs. 3.12 ± 0.15 years, respectively, $p = 0.13$) or in the proportion of female children in the two groups (47.97% vs. 57.14%, respectively, $p = .14$).

Descriptive information on the participants is shown in Table 1. The children with PCE were significantly smaller in terms of birth weight, occipital-frontal head circumference, and gestational age calculated using the methods outlined by Dubowitz et al.¹⁹ While the mean between-group difference in gestational age was significant, both groups were born at full-term (>37 weeks) on average. The biological mothers of the children with PCE used significantly more tobacco, alcohol, and marijuana; were significantly older; entered prenatal care later; and had higher pregnancy risk scores as measured by the Hobel Obstetric Risk Scale²⁰ than the mothers of the children in the matched control group. All of these groups differences parallel those found in the original cohort.¹⁷ Table 1 also illustrates that the children with PCE were much more likely to have changed caregivers at least once since birth compared to the children with no PCE. Only 52% of the children with PCE, compared to 95% of the non-cocaine exposed children, were living with their biological mothers at the time of the 3-year assessment.

Measures

Prenatal Drug Exposure—Average daily use for tobacco cigarettes, absolute ounces of alcohol, and marijuana joints were calculated using the usual daily amounts reported by each woman during the pregnancy and prorated by the frequency of occasions when more or less than usual use was reported. Average cocaine use during pregnancy was calculated as a ratio of the number of weeks of reported cocaine use divided by the total number of weeks of each woman's individual gestation plus the 12 weeks prior to gestation. Previous work by our group has determined that the frequency of cocaine use for the 12 weeks prior to pregnancy and throughout pregnancy rather than the amount of cocaine use during pregnancy is more significantly related to typical birth outcomes of neonates with PCE in our sample.²¹

Caregiver and Environment Measures—Caregiver depression was assessed using Center for Epidemiologic Studies-Depression (CES-D) scale.²² The CES-D is a widely used 20-item self-report screening measure used to assess symptoms associated with depression, including changes in mood, somatic complaints, and interpersonal problems. Items are rated in terms of frequency during the past six months on a four-point Likert scale ranging from 0 (*rarely or none of the time*) to 3 (*most of the time*). A total score is derived from the sum of the 20 items and a cutoff score of greater than or equal to 16 is considered high and suggestive of a depressive disorder. High internal consistency and adequate test-retest reliability for the CES-D were demonstrated in the early studies associated with the development of the scale.²²⁻²⁴ The validity of the CES-D has been established by more than 30 years of research using community-dwelling and patient groups in the United States and abroad. A recent study replicated the original factor structure of the CES-D in a sample of low socioeconomic status African Americans.²⁵

Assessment of the child's caregiving environment was made using the Home Observation for Measurement of the Environment (HOME).²⁶ The HOME is a screening measure that assesses factors related to nurturance and stimulation in a child's home environment that are believed to be important for cognitive development. The HOME was designed as an alternative to sociodemographic factors for identifying children at "high risk" for intellectual/academic problems. Scores are based on both the observer's visual inspection of the home and self-report of the child's primary caregiver obtained through a semi-structured interview during a 45- to 90-minute home visit. The preschool version of the HOME (later renamed the Early Childhood version) is designed for use with children ages 3 to 5 years and contains 55 items clustered in eight rationally derived scales: Learning Stimulation, Language Stimulation, Physical Environment, Warmth and Acceptance, Academic Stimulation, Modeling of Social Maturity, Variety in Experience, and Physical Punishment. A total score is derived from the sum of the eight scales. The HOME has demonstrated acceptable reliability and validity and has been validated in samples of African American children.^{27,28}

Child Behavior Outcome Measures—Caregiver report of child behavior problems was obtained using the CPRS-48¹⁴ and the ECBI.¹⁵ The CPRS-48 is a parent report measure for assessing the problem behaviors of children ages 3 to 17 years and represents a shorter version of the original 93-item CPRS developed using a different normative sample.²⁹ Instructions indicate that the rating should reflect how much the parent thinks his/her child has been bothered by each item during the past month. Parents rate each item on a Likert-type scale that ranges from 0 (*not at all*) to 3 (*very much*). Scores are derived for five scales (Conduct Problem, Learning Problem, Psychosomatic, Impulsive-Hyperactive, and Anxiety) and one index (Hyperactivity Index). The Hyperactivity Index is composed of three independent items and seven items drawn from the Conduct Problem, Learning Problem, and Impulsive-Hyperactive scales. The scores for each scale and the index are converted to *t*-scores with separate normative profiles for sex and age groupings. Factor analyses of the CPRS-48 revealed two distinct factors for conduct problems and the impulsive-hyperactive behaviors, which were used in the current study.¹⁴ The CPRS-48 has demonstrated good discriminant validity, test-retest and interrater reliability, long-term stability, and sensitivity to treatment effects.³⁰⁻³⁴ No data are available regarding the appropriateness of the CPRS-48 for African American children; however, scores were independent of SES in the normative sample.²⁹

The ECBI is a 36-item parent report measure of disruptive behavior problems for children ages 2 to 16. Behaviors are assessed on both an Intensity Scale and a Problem Scale. The Intensity scale measures the frequency of behaviors on a scale from 1 (*never*) to 7 (*always*), providing a measure of the severity of disruptive behavior. The Problem scale is a *yes-no* scale that assesses whether or not the parent finds each behavior problematic, providing a measure of parental distress related to the child's behavior. The total Intensity score is a sum of the ratings for each behavior and ranges from 36 to 252. The total Problem score is the number of behaviors indicated as problematic (i.e., circled *yes*) and ranges from 0 to 36. Intensity scores ≥ 131 and Problem scores ≥ 15 are considered clinically significant. The ECBI has been well validated in both clinic-referred and non-referred samples and has demonstrated good reliability and validity.^{15,35-40} The original ECBI was not validated in African American samples; however, the newer version published in 1999 does not differ substantially from the older version used in this study and is reported to have an internal consistency of .97 for the African American subgroup in the standardization sample.⁴¹

Data Analyses

Prior to any statistical analyses, the data were inspected for violations of normality. All variables used in these analyses were transformed using a square root procedure. Missing data for the CES-D and HOME, which represented less than 1% of the total data, were imputed using the Markov Chain Monte Carlo method.⁴² SAS 8.2⁴³ was used to conduct data screening and all statistical analyses. The criterion for significance tests was set at $\alpha = .05$, two-tailed. Group comparisons were made using the Wilcoxon rank sum test for continuous data and the chi-square statistic for categorical data.

Structural equation modeling (SEM) was conducted using the PROC CALIS procedure in SAS, which uses the maximum likelihood estimation method for estimating free parameters. All models were estimated in covariance metric. Power analysis based on the sample size and degrees of freedom⁴⁴ indicated that the power of the entire theoretical model to detect significant relationships between the variables is greater than 0.80. Model fit was evaluated using Bentler's comparative fit index (CFI),⁴⁵ McDonald's Centrality Index (MCI),⁴⁶ the standardized root-mean-square-residual (RMR) and the root-mean-square error of approximation (RMSEA). The recommended cutoff values for good fit are $>.95$ for the CFI, $>.90$ for the MCI, $<.08$ for the RMR, and $<.06$ for the RMSEA.⁴⁷ Using multiple indicators of model fit is recommended to ensure reasonable rejection rates for various types of misspecified models.⁴⁷⁻⁴⁹

SEM is a powerful statistical technique that involves multiple regression analyses of factors. Factors, called latent constructs, are derived from the reliable shared variance of one or more observed indicators and thus are theoretically free of measurement error. SEM allows for the examination of complex relationships between multiple continuous and categorical independent variables and multiple continuous and categorical dependent variables. After specification of a theoretical model, which is a type of confirmatory factor analysis, SEM can be used to test a model of relationships between observed variables and latent constructs, test specific hypotheses about a model, and modify an existing model.⁵⁰

The structure of the model predicting child behavior problems was based on theoretical grounds as well as a review of the literature and previous research by our group. We hypothesized that PCE would have a direct negative effect on child behavior problems and a negative indirect effect through relationships with maternal/caregiver depression and the quality of the child's environment. Maternal/caregiver depression and the quality of the child's home environment when the child was age 3 were expected to be direct predictors of child behavior problems. Maternal/caregiver depression was also expected to have an indirect effect on reported child behavior problems through its relationship with the quality of the child's home environment. Child sex was included in the model as sex differences in the prevalence of disruptive behavior problems, with boys scoring significantly higher than girls, have been found in normative samples for parent rating scales^{51,52} and in community and epidemiological samples.⁵³⁻⁵⁶ Prenatal exposure to alcohol, tobacco, and marijuana was controlled by creating an Other Substance Use latent factor that was allowed to covary with PCE.

RESULTS

Group Comparisons of PCE and Non-Exposed Children

Comparisons were made between the cocaine-exposed and non-exposed groups for the 5 psychosocial measures in the study. As shown in Table 2, no significant differences were found for scores on the CES-D, HOME, CPRS-48 Conduct Problems, CPRS-48 Impulsive-Hyperactive Problems or the ECBI Intensity scale (p 's $>.05$). There was a non-significant trend for higher CES-D scores among the caregivers of non-exposed children as compared

to the PCE group ($p = .06$). Of note, the mean CES-D score for both groups fell in the clinically significant range (score ≥ 16). Similarly, the mean scores for the child behavior problem measures were around one standard deviation above the normative sample means. Table 3 shows the percentage of children in each group whose scores were above the cutoff for clinical significance for each measure. While there were no significant between-group differences, the proportion of both cocaine-exposed and non-exposed children who had CPRS-48 and ECBI scores in the clinically significant range was remarkably high compared to the expected incidence for conduct problems in the general population. Between 29% to 33% of the children with PCE and 26% to 42% of the non-exposed were identified as having behavior problems in the clinically significant range.

Structural Equation Modeling

An initial model that included a dichotomous placement variable (with or not with biological mother at age 3) did not meet criteria for good fit. When this variable was excluded, both the measurement model and our proposed theoretical (structural equations) model displayed acceptable fit (Table 4). The measurement model, by definition, allows for all possible correlations between each of the observed and latent variables in the model and serves as a baseline model. Our proposed theoretical model specifies particular relationships between the observed and latent variables in the model, thereby reducing the number of parameters that must be estimated. The chi-square difference test between the theoretical and measurement models was non-significant, indicating that the theoretical model adequately accounted for the underlying structure of the observed data as well as the measurement model: $\chi^2(6, N = 256) = 3.69, p > .05$. Table 5 shows the path coefficients, standard errors, and t values for the structural equation model. Figure 1 shows the structural equation model with the standardized path coefficients and factor loadings of the indicators on their respective latent constructs. The only significant predictor of Child Behavior Problems at age 3 was the CES-D ($\beta = .41$). The CES-D was also a significant predictor of the HOME ($\beta = -.18$), but contrary to expectations the HOME was not significantly related to Child Behavior Problems. PCE and child sex were also not significant direct predictors of Child Behavior Problems. Although the CES-D was the only significant predictor of caregiver-reported behavior problems, the overall model explained 21% of the variance in Child Behavior Problems at age 3 in this combined sample of children with and without PCE.

Post Hoc Comparisons Using Cocaine Exposure and Child Placement Status

Post hoc analyses were conducted to understand the role that cocaine exposure (exposed vs. non-exposed) and placement status (with vs. not with biological mother at age 3) may have played in the relationship between the CES-D and Child Behavior Problems found in the SEM. A one-way ANOVA by group was used to compare ratings of biological mothers of non-exposed children ($n = 127$), biological mothers who retained custody of their children with PCE ($n = 63$) and non-maternal caregivers who had assumed custody of children with PCE ($n = 60$). There were too few children in the control group in out-of-home placement to make meaningful comparisons. Results revealed a significant effect of group for the CES-D [$F(2, 249) = 20.55, p < .0001$], the CPRS-48 Conduct Problems scale [$F(2, 249) = 3.72, p = .03$] and the ECBI Intensity Scale [$F(2, 249) = 3.39, p = .04$] but not for the CPRS-48 Impulsive-Hyperactive scale [$F(2, 249) = .70, p = .50$]. As shown in Table 6, comparisons using the Ryan-Einot-Gabriel-Welsch Multiple Range test, which balances protection against both Type I and Type II errors, found that the significant group differences for the CES-D, the CPRS-48 Conduct Problems scale and the ECBI Intensity scale were due to significantly lower mean ratings given by non-maternal caregivers for the cocaine-exposed children in their custody as compared to the other two groups. There were no significant mean differences between the non-exposed and cocaine-exposed groups who were with their

biological mothers for any of the measures. For both groups of biological mothers, the mean CES-D score was well above the cutoff for clinical significance (score ≥ 16).

Table 7 shows the proportion of children rated as having clinically significant behavior problems by cocaine exposure and placement status. Chi-square analyses indicated a significant effect of group for the CPRS-48 Conduct Problems scale [$\chi^2(2) = 5.90, p = .05$] and the ECBI Intensity scale [$\chi^2(2) = 7.22, p = .03$] but not for CPRS-48 Impulsive-Hyperactive scale [$\chi^2(2) = .09, p = .96$]. As shown in Table 7, pairwise comparisons with Bonferroni correction revealed that the group difference for the CPRS-48 Conduct Problems scale was due to the significantly lower ratings given by non-maternal caregivers of children with PCE as compared to the biological mothers of the non-exposed children. For the ECBI Intensity scale, the group difference was due to significantly lower ratings given by the non-maternal caregivers of children with PCE as compared to the biological mothers of children with PCE. There were no significant differences between the biological mothers of children with PCE and the biological mothers of non-exposed children in the proportion of children rated as having clinically significant problems on any of the three measures. For all three measures, non-maternal caregivers of children with PCE rated the lowest proportion of children as having clinically significant behavior problems. All three groups of mothers/caregivers rated much larger proportions of their children, regardless of cocaine exposure status, as having clinically significant behavior problems (17% to 43%) across all measures than what would be expected based on prevalence of disruptive behavior disorders in the general population.

DISCUSSION

The non-association between PCE and caregiver-reported child behavior problems in the current study is consistent with the growing literature in this area. Negative findings have been reported for five studies, all of which used the CBCL to assess behavior problems.^{4,5,11,57,58} Using scales from the CPRS-48 and ECBI, two parent report measures specifically designed to assess disruptive behaviors and which might be expected to be more sensitive than an omnibus measure like the CBCL, yielded no significant differences between children with PCE and non-exposed children at age 3. Both groups, on average, obtained scores within the normal range on the behavior ratings scales used.

More studies to date have shown that scores on standardized parent assessments of child behavior for children with PCE are more related to maternal psychological functioning than to *in utero* cocaine exposure.^{4,5,7,11} In the current study, concurrent maternal/caregiver depression as measured by the CES-D was the only significant predictor of reported child behavior problems in a model that included prenatal drug exposure, child sex, and the quality of the child's home environment. Notably, the mean CES-D scores did not differ significantly between the mothers/caregivers of the cocaine-exposed and non-exposed groups as a whole. However, when biological mothers of the cocaine-exposed and non-exposed children were compared to the non-maternal caregivers of cocaine-exposed children, the two groups of biological mothers did not differ from each other but had significantly higher mean CES-D scores than the non-maternal caregivers. Further, the mean CES-D scores for both groups of biological mothers were well above the recommended clinical cutoff used to screen for depression. A similar pattern was found for the three behavior ratings scales used. As a whole, no differences were found between the cocaine-exposed and non-exposed groups for mean behavior problem scores or proportion of children rated as having clinically significant behavior problems. When biological mothers of non-exposed and cocaine-exposed children were compared to the non-maternal caregivers of cocaine-exposed children, differences emerged for 2 of 3 the behavior ratings scales used with significantly higher ratings and larger proportion of children as having clinically

significant behavior problems reported by both groups of biological mothers. These findings suggest that the relationship between maternal depression and reported child behavior problems was present in both the group of cocaine-using mothers and the group of mothers/caregivers of non-exposed children.

It is well established that children of depressed mothers are at a greater risk for emotional and behavioral problems than children whose mothers are not depressed.⁵⁹⁻⁶² Longitudinal studies have revealed that chronic maternal depression from the time of a child's birth, as well as concurrent reports of maternal depression, are associated with more child behavior problems.^{63,64} Based on the high mean levels of depressive symptoms reported by both groups of caregivers in our study, non-exposed and cocaine-exposed children appear to be at risk for poor behavioral outcomes. We suspect that maternal/caregiver depression in the current study is related to the considerable poverty reported by our participants and the stressors associated with a lack of financial and other resources.

Remarkably high rates of clinically significant behavior problems were reported for both the cocaine-exposed and the non-exposed children, compared to the expected incidence for disruptive behavior disorders in the general population. Using standard clinical cutoffs, rates ranged from 29% to 33% of the children with PCE and 26% to 42% of non-exposed children depending on the measure. These results are, however, consistent with the high rates of reported behavior problems (33–34%) in a mixed cohort similar to ours.⁶ Considering the well-known negative reporting bias shown by depressed mothers in general,⁶⁵ the depressed mothers in our sample may have over-reported the number and/or severity of behavior problems in their children. Low literacy may also have influenced how participants responded to the questionnaires. The mothers and caregivers in our study had, on average, less than a high school education and may have misunderstood the questionnaire items, although they were read to the participants by the interviewers in an attempt to avoid literacy problems. The fact that non-maternal caregivers, whose depressive symptoms scores were in the normal range on average, also reported high rates of behavior problems that were not significantly different from that of the biological mothers on 2 of the 3 behavior scales used lends some support for this idea.

Despite the methodological strengths of this study, including prospective participant enrollment, a demographically matched comparison group, a large sample size, and high retention rate, a number of limitations should be considered when interpreting the results. One important limitation is the use of maternal/caregiver report to assess child behavior problems. Observational data and preschool teacher reports could have helped to confirm the presence or absence and severity of disruptive behavior problems in our sample and enriched our ability to determine how a variety of environmental factors affect behavioral functioning. Conclusions drawn from the results are also limited to the externalizing behavior problems assessed by the measures used. The model we tested for predicting child behavior problems did not include all the possible predictors and confounders that could explain behavior problems in children with PCE, such as current maternal substance use. An attempt to include out-of-home placement for the children with PCE, almost half of whom were not in the custody of their biological mothers by age 3, in our analyses was unsuccessful. In addition, the measures of maternal/caregiver depression and the quality of the home environment were cross-sectional and concurrent with the behavior problems ratings. Characteristics related to the nature of our sample – primarily poor, rural, African American children who were, on average, born full-term and of normal birth weight to generally healthy mothers over the age of 18 – also place some limitations on generalizing these results to other populations.

This study's contributions include use of parent report measures not previously reported in the literature, comparisons between biological mothers and non-maternal caregivers of children with PCE on measures of psychological functioning and their ratings of child behavior problems, and use of structural equation modeling to evaluate a model of behavior problems in children with PCE. The results add to the growing body of evidence suggesting that expectations of increased behavioral dysregulation in children with PCE based on the reported effects of PCE on monoaminergic systems in the brain^{66,67} are generally not warranted. Our findings highlight the need for clinicians to consider screening all parents, substance-abusing or not, for previous and current depressive symptoms when they present their children for evaluation and/or treatment of disruptive behavior problems. Priorities for future research related to the behavioral functioning of children with PCE should include examining developmental trajectories that take into account postnatal factors specific to this population, such as chronic maternal substance abuse and depression, caregiver changes, frequent household moves and school changes. Investigations focusing on non-maternal caregivers, including variables such as kinship care versus non-kinship care and the timing and duration of care, could yield important information related to developmental outcomes of children with PCE. Finally, considering that on average children with PCE have not been identified as having significant disruptive behavior problems in several studies, researchers should seek to identify the possible protective factors, such as academic achievement and support from extended family, that promote resilience in this group of children who are generally considered to be at risk for poor outcomes.

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FIGURE 1.

Structural equation model predicting child behavior problems at age 3, $\chi^2 = 50.68$, $df = 30$, $p = .01$, CFI = .94, MCI = .06, RMR = .03, RMSEA = .05. CES-D was the only significant predictor of Child Behavior Problems, explaining 21% of the variance. Solid lines represent significant paths while dashed lines indicate non-significant paths. A line with two arrows indicates variables allowed to covary. The path between prenatal cocaine exposure and the HOME was non-significant ($\beta = -.04$). CES-D, Center for Epidemiological Studies-Depression; CPRS-48, 48-item Conners' Parent Rating Scale; ECBI, Eyberg Child Behavior Inventory, HOME, Home Observation for Measurement of the Environment.

Table 1

Sample Description

Characteristic	Cocaine-exposed (n = 123) Mean (SD)	Non-exposed (n = 133) Mean (SD)	p Value
Child			
Gestational age (wks)	38.54 (2.05)	39.05 (1.92)	.03
Birth weight (g)	3042.98 (606.75)	3235.40 (592.62)	.01
Head circumference at birth (cm)	33.48 (1.96)	34.39 (1.49)	<.005
Number of caregiver changes since birth	.54 (.94)	.09 (.38)	<.0001
With biological mother at age 3 (%)	51.64	95.49	<.0001
Mother			
Cigarettes during pregnancy (no./day)	8.40 (9.11)	2.20 (6.07)	<.0001
Alcohol during pregnancy (oz absolute/day)	.23 (.38)	.02 (.06)	<.0001
Marijuana during pregnancy (joints/day)	.18 (1.13)	.01 (.12)	.0001
Age at delivery (years)	27.95 (4.77)	24.00 (5.65)	<.0001
Week entered prenatal care *	15.28 (7.76)	12.43 (7.37)	<.005
Hobel Prenatal Risk †	55.17 (18.81)	43.12 (18.69)	.0001
Education (years)	11.38 (1.47)	11.34 (1.04)	NS
Socio-economic status	4.70 (.54)	4.74 (.52)	NS
African American (%)	82.11	81.95	NS
Multiparous (%)	86.99	87.97	NS

NS, not significant. Comparisons were made using the Wilcoxon rank sum test for continuous variable and the chi-squared statistics for categorical variables.

* n = 111 for cocaine-exposed, n = 129 for non-exposed due to missing data.

† n = 119 for cocaine-exposed, n = 130 for non-exposed due to missing data.

Table 2

Caregiver Depression, Home Environment and Child Behavior Problems Variable by Drug Exposure Group

Variable	Cocaine-exposed (n = 123) Mean (SD)	Non-exposed (n = 133) Mean (SD)	p Value
CES-D	20.20 (10.66)	22.37 (9.38)	.06
HOME	38.76 (8.67)	38.58 (8.05)	.83
CPRS-48 Conduct problems scale	58.23 (13.46)	61.11 (14.50)	.11
CPRS-48 Impulsive-hyperactive scale	59.59 (9.62)	60.94 (9.38)	.33
ECBI Intensity scale	111.36 (29.18)	116.74 (30.60)	.19

CES-D, Center for Epidemiological Studies-Depression; CPRS-48, Conners' Parent Rating Scale; ECBI, Eyberg Child Behavior Inventory; HOME, Home Observation for Measurement of the Environment.

Table 3

Proportion of Children with Behavior Problems in the Clinical Range by Drug Exposure Group

T score	Cocaine-exposed (n = 122)	Non-exposed (n = 133)	p value
CPRS-48 Conduct Problems ≥ 65	32.52%	42.11%	.11
CPRS-48 Impulsive-Hyperactive ≥ 65	32.52%	34.59%	.73
ECBI Intensity ≥ 131	27.64%	30.08%	.67

CPRS-48, Conners' Parent Rating Scale; ECBI, Eyberg Child Behavior Inventory.

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Table 5

Path Coefficients, Standard Errors, and T-Values for Structural Equation Model

	Unstandardized PathCoefficient	Standard Error	T Value	Standardized PathCoefficient
Child Behavior Problems				
Prenatal cocaine exposure	-.18	.20	-.90	-.06
Child sex	-.16	.14	-1.15	-.07
CES-D	.37	.06	5.90	.41
HOME	-.16	.10	-1.57	-.10
CES-D				
Prenatal cocaine exposure	-.39	.21	-1.87	-.12
HOME				
Prenatal cocaine exposure	-.07	.12	-.58	-.04
CES-D	-.10	.04	-2.84	-.18
Prenatal cocaine exposure				
Other prenatal drug exposure	.29	.03	10.42	.80

CES-D, Center for Epidemiological Studies-Depression, HOME, Home Observation for Measurement of the Environment.

Table 6

Caregiver Depression and Child Behavior Problems by Cocaine Exposure and Placement Status

	Non-exposed with Biological Mother (n = 127)	Cocaine-exposed with Biological Mother (n = 63)	Cocaine-exposed with Other Caregiver (n = 60)
Variable	Mean (SD)	Mean (SD)	Mean (SD)
CES-D	22.80 _a (9.22)	25.13 _a (9.51)	15.02 _b (9.30)
CPRS-48 Conduct problems scale	61.40 _a (14.38)	60.71 _a (13.06)	55.62 _b (13.48)
CPRS-48 Impulsive-hyperactive scale	60.91 _a (9.51)	59.97 _a (9.56)	59.18 _a (9.74)
ECBI Intensity scale	117.22 _a (31.04)	116.87 _a (26.94)	105.57 _b (30.51)

CES-D, Center for Epidemiological Studies-Depression; CPRS-48, Conners' Parent Rating Scale; ECBI, Eyberg Child Behavior Inventory. Means in the same row that do not share subscripts differ at $p < .05$ using the Ryan-Einot-Gabriel-Welsch Multiple Range test.

Table 7

Proportion of Children with Behavior Problems in the Clinical Range by Cocaine Exposure and Placement Status

	Non-exposed with Biological Mother (n = 127)	Cocaine-exposed with Biological Mother (n = 63)	Cocaine-exposed with Other Caregiver (n = 60)
T-score	%	%	%
CPRS-48 Conduct problems $\geq$ 65	43.31 _a	39.68 _{a,b}	25.00 _b
CPRS-48 Impulsive-hyperactive $\geq$ 65	33.86 _a	33.33 _a	31.67 _a
ECBI Intensity $\geq$ 131	31.50 _{a,b}	38.10 _a	16.67 _b

CPRS-48, Conners' Parent Rating Scale; ECBI, Eyberg Child Behavior Inventory. Values in the same row that do not share subscripts differ at $p < .02$ using the chi-square test.