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Maternal Cocaine Use: Estimated Effects on Mother-Child Play Interactions in the Preschool Period

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Abstract

The study objective was to evaluate the quality of parent-child interactions in preschool-aged children exposed prenatally to cocaine. African-American mothers and their full-term newborns ($n = 343$) were enrolled prospectively at birth and classified as either prenatally cocaine-exposed ($n = 157$) or non-cocaine-exposed ($n = 186$) on the basis of maternal self-report and bioassays. Follow-up evaluations at 3 years of age (mean age, 40 mo) included a videotaped dyadic play session and maternal interviews to assess ongoing drug use and maternal psychological distress. Play interactions were coded using a modified version of Egeland et al's Teaching Task coding scheme. Regression analyses indicated cocaine-associated deficits in mother-child interaction, even with statistical adjustment for multiple suspected influences on interaction dynamics. Mother-child interactions were most impaired in cocaine-exposed dyads when the mother continued to report cocaine use at the 3-year follow-up. Multivariate profile analysis of the Egeland interaction subscales indicated greater maternal intrusiveness and hostility, poorer quality of instruction, lower maternal confidence, and diminished child persistence in the cocaine-exposed dyads.

Keywords

cocaine-exposed; parent-child interaction; maternal psychological functioning

This article investigates the degree to which maternal cocaine use might have an adverse impact on mother-child interaction during the preschool years, with videotaped mother-child interactions measured using the Egeland et al¹ coding system. This subject matter is topical even though there has been an abatement and plateau of the U.S. cocaine epidemic since the mid-1980s.²⁻³ The most recent epidemiological evidence continues to show a sizable percentage of women, many of whom are mothers, are involved in cocaine use and struggle with cocaine-related problems and dependence.³ Maternal substance use has been associated with deficits in parental functioning in numerous studies.⁴⁻⁸ Cocaine/polydrug-using mothers have been described as more passive and disengaged in interactions with their newborns than nondrug-using mothers,⁷ and methadone/polydrug-using mothers have been characterized as less responsive and less encouraging of their infants than nondrug-using mothers.⁵ Krauss⁸ reported that cocaine-using mothers were less emotionally accessible and developmentally appropriate with their preschool-aged children than were noncocaine-using mothers, whereas Bauman and Dougherty⁴ found that drug-using mothers were less likely to use positive reinforcement and more likely to threaten physical discipline than were a comparison group of nondrug-using mothers. Finally, Wasserman and Leventhal⁹ found a higher incidence of foster-care placements and substantiated allegations of physical abuse, sexual abuse, and neglect among cocaine-exposed 2 year olds compared with nondrug-exposed 2 year olds. These studies indicate that mothers who use drugs have more difficulty relating to and interacting with their young children. Moreover, women who use cocaine and other drugs during pregnancy often continue to use long after delivery, with postnatal maternal substance use associated with a number of other environmental risk factors that may impact parent-child interactions.¹⁰

Maternal drug use, although frequently associated with poor parenting outcomes, is not invariably related to deficits in parenting skills. Johnson and Rosen¹¹ assessed maternal responsivity among a cohort of mothers with varying degrees of substance use and found no association between the severity of prenatal drug use and maternal responsivity during free-play. Neuspiel and others¹² observed no differences between cocaine-using and comparison mothers in sensitivity, social-emotional and cognitive growth fostering, and response to infant distress on a measure of maternal behavior during infant feeding. Similarly, Black et al¹³ reported no differences between cocaine/polydrug-using mothers and comparison mothers on a measure of maternal nurturance during an unstructured play interaction. Jeremy and Bernstein¹⁴ suggested that maternal drug use alone may be a poor predictor of parent-child interaction skill, especially when indices of maternal psychological functioning and psychosocial resources are taken into account. These factors alone or in combination with maternal substance abuse may better explain the impact of prenatal drug use on mother-child relationship outcomes.¹⁵

The association between maternal substance use and other mental health disorders (i.e., depression, anxiety, personality disorders, and posttraumatic stress disorder) is well supported in the literature.¹⁶⁻²¹ Pregnant, drug-using women have been found to have higher levels of depressive symptoms than pregnant, nondrug-using women.²¹ Postpartum, drug-using mothers report higher levels of depression and more numerous posttraumatic stress disorder symptoms than do postpartum, nondrug-using mothers.¹⁹ Singer et al²⁰ found significantly more paranoid and phobic anxiety symptoms in postpartum drug-using mothers compared with postpartum drug-free mothers. Gaulier et al¹⁸ reported a relatively high incidence of personality disorders among a small sample of drug-using women recruited from an inpatient substance abuse ward and a perinatal addictions program. Although not using a control group, Chavkin et al¹⁷ nonetheless reported that as many as 58% of treatment-seeking, drug-using mothers had a prior history of psychological problems. Other investigators have drawn similar conclusions and have noted that mothers with both drug and psychological problems have greater difficulty relating to and responding to their infants than do mothers with drug problems alone.²² Two

recently published studies indicate that prenatal symptoms of depression and paranoia in drug-using women are associated with impairments in parental functioning 6 months later.^{14,23}

Although there is some convincing evidence that mothers who use drugs are at risk for early difficulties in parental functioning, few studies have investigated whether maternal drug use is associated with parent-child interaction difficulties once other risk factors are taken into account.²⁴ Broadening our knowledge of how maternal substance use and psychological functioning might impact the developing mother-child relationship will facilitate improved understanding of the dynamics of high-risk familial environments. Such an understanding is likely to improve the ability to identify children and mothers affected by maternal substance use and design appropriate clinical interventions to improve long-term outcomes. The current study represents an attempt to evaluate whether maternal cocaine use during and after pregnancy is associated with parent-child interactions during the early preschool years, apart from other important psychosocial risk factors such as maternal psychological functioning.

Researchers frequently use observational methodologies in investigations of parent-child relationship dynamics. One approach is to assess discrete parenting behaviors, such as the number of maternal positive statements,²⁵ whereas another approach is to assess more global dimensions of parenting such as supportiveness, sensitivity, and authoritarianism.²⁶ The current investigation assesses parent-child interactions with a modified version of Egeland et al's¹ Teaching Task coding scheme. This system derives from an attachment-based theoretical model whereby secure mother-child dyads negotiate the parameters around obstacles in an effort to achieve common goals, reciprocally reinforce one another both verbally and nonverbally, and successfully balance the needs of the child with the practical constraints on the caregiver.

The coding system of Egeland et al¹ has been used with minority and high-risk mother-child dyads.^{27,28} For example, in a cohort of mothers characterized as at risk because of low socioeconomic status (SES), low maternal educational attainment, single parenthood, and unstable living conditions, indices of maternal psychosocial stress were negatively associated with the quality of mother-child interactions involving preschool-aged girls and weakly associated with the quality of mother-child interactions involving preschool-aged boys.²⁹

This study's objective is to estimate the hypothesized relationship between prenatal cocaine use and maternal-child interactions during the preschool years. Additional research questions include whether recent maternal cocaine use and psychological distress are independently associated with mother-child interactions after prenatal cocaine use has been considered. Multiple regression models are used to evaluate these research questions with the inclusion of additional potential confounding influences such as prenatal exposure to other drugs and important maternal and child characteristics measured at birth and 3-year follow-up.

METHODS

Study Design

As part of a larger epidemiological study of in utero substance exposure, 476 mother-infant dyads were enrolled prospectively in a follow-up cohort to evaluate the effects of prenatal cocaine exposure on long-term developmental outcome. Subjects were recruited during the immediate postpartum period from the delivery service of the University of Miami School of Medicine Jackson Memorial Medical Center. The overall study design and recruitment of the follow-up cohort for the longitudinal Miami Prenatal Cocaine Study (PCS) have been detailed in a separate report³⁰ and are briefly summarized below. The study was approved by the institutional review board and conducted under a federal Department of Health and Human Services (DHHS) Certificate of Confidentiality.

Study Participants

The original follow-up sample for the Miami PCS was recruited between November 1990 and July 1993 and was restricted to mothers of full-term infants (gestational age ≥ 37 completed weeks) who were self-identified as African-American women born in the United States. Eligibility was also restricted with respect to low socioeconomic status (SES) and inner-city residence as determined by maternal self-report to improve statistical power and covariate control. Infants with maternal HIV/AIDS infection; prenatal exposure to opiates, methadone, amphetamines, barbiturates, benzodiazepines, or phencyclidine; major congenital malformations; chromosomal aberration; or disseminated congenital infection were excluded. Prenatal cocaine exposure was assessed by maternal self-report of cocaine use during pregnancy and by biological markers, including maternal urine, infant urine, and meconium. Assignment to the cocaine group was based on documentation of one or more of the following: positive maternal self-report of cocaine use during pregnancy and/or least one confirmed cocaine-positive biological marker. Of the 253 identified cocaine-exposed infants in the follow-up cohort, 40 cases (16%) were identified as cocaine-exposed solely on the basis of positive maternal self-report. In 80 cases (32%) the mother denied cocaine use during pregnancy, but one or more biological markers were cocaine positive. The original follow-up sample consisted of 253 cocaine-exposed infants (with or without concomitant use of alcohol, tobacco, or marijuana) and 223 non-cocaine-exposed infants, of whom 147 were drug-free and 76 were exposed to varying combinations of alcohol, tobacco, or marijuana.

The current report focuses on data collected at the preschool assessment visit at 3 years of age. From the 476 mother-infant pairs initially enrolled at delivery, 439 (92%) children returned for the preschool follow-up exam. From the 439 children who returned to the preschool assessment, data analyses and conclusions are limited to the subset of infants ($n = 345$) who returned for the preschool assessment in the care of the biological mother and for whom a mother-child interaction videotape was obtained and rated. Excluded from the current analyses were 89 children who were accompanied to the assessment by an alternate caregiver such as a grandmother or maternal aunt, five children who completed mother-child interactions with their mothers but whose videotapes were either corrupted or inaudible, and 37 remaining children who did not attend the preschool visit for varied reasons ($n = 37$; 21 moved, 7 refused, 6 never showed, 2 died, 1 lost to follow-up).

Comparisons of baseline demographic variables between the subsample under study ($n = 343$) and those from the original cohort who were not included as previously described ($n = 133$; 96 cocaine-exposed, 37 non-cocaine-exposed) revealed no differences in infant sex, gestational age, maternal education, employment, marital status, or self-reported amount of drug use during pregnancy (cocaine, marijuana, alcohol, and tobacco). Mothers included in the present report were younger at delivery, were more likely to be first-time mothers, and had attended a greater number of prenatal visits than mothers who were not included. In separate analyses within the cocaine-exposed and comparison groups on the same set of variables, no differences were noted with the exception of more prenatal care visits and higher educational attainment for mothers in the cocaine-exposed group included in the study when compared with those not included. Within the comparison group, mothers in the study reported attending a greater number of prenatal visits than did the mothers not in the study cohort. Of the 89 children who attended the preschool visit with an alternate care provider, 85% were from the cocaine-exposed group; however, maternal self-reported amount of cocaine use during pregnancy was similar within the cocaine-exposed group between mothers included and not included in the present report.

Prenatal Drug Exposure Measures

Maternal Interview—A structured, standardized interview was conducted by trained research staff within the first 36 hours postpartum to ascertain maternal prenatal substance use and additional demographic information. Prenatal drug-use questions by trimester included the number of weeks used, most days per week, fewest days per week, usual days per week, and usual dose per day. Dosage was recorded in the number of cigarettes smoked per day, number of marijuana joints smoked per day, and number of drinks of beer, wine, or hard liquor. Standardized definitions³¹ were used for determining one-drink units for each type of alcohol (beer 12 oz, wine 5 oz, and liquor 1.5 oz). Cocaine dosage was recorded in the number of rocks or lines used per day. Amount exposure composites for total pregnancy were calculated for each drug by multiplying the number of weeks used by the usual days per week and the usual dose per day. In the present report, self-reported alcohol, tobacco, and marijuana were analyzed as covariates using these pregnancy composites. Prenatal cocaine exposure was evaluated as a dichotomous grouping indicator, including information from both self-report and biological markers.

Biological Markers (Meconium and Urine)—Screening of maternal and infant urine and meconium for cocaine Cocaine and Maternal-Child Play Interaction 193 metabolite (benzoylecgonine) was performed by EMIT® (Syva Corporation, Palo Alto, CA), at a cutoff of 150 ng/mL urine and 150 ng/g meconium, respectively, and cocaine-positive specimens were confirmed by gas chromatography/ mass spectrometry.³² Urine specimens were also assayed by EMIT® for marijuana (cannabinoids), opiates, amphetamines, barbiturates, benzodiazepines, and phencyclidine. Meconium specimens were also assayed by EMIT® for marijuana and opiates.

Infant Birth Measures

Birth weight was recorded immediately after delivery. Within the first 36 hours, research personnel blinded to exposure status measured occipital-frontal head circumference and recumbent crown-heel birth length and performed the Ballard³³ gestational age assessment. Pertinent additional maternal and infant medical and demographic information were obtained by medical record review.

Parent-Child Interactions

Teaching Tasks Coding Scheme—The Teaching Tasks coding scheme,¹ conceptualized from Bowlby's³⁴ theory of attachment, is an observation-based rating scale that assesses the quality of mother-child interactions. Initially designed to accompany a 25-minute, standardized, parent-child interaction labeled the "Teaching Tasks," the coding scheme has also been used to evaluate dyadic interactions in less-structured play settings (Nancy Weinfield, personal communication, 1997). The coding scheme contains 14 rating scales. Five scales assess maternal behavior: supportive presence, intrusiveness, hostility, quality of instruction, and confidence. Seven scales assess the child's behavior: persistence, enthusiasm, negativity, compliance, experience of the session, affect to mother, and avoidance of mother. Two scales assess the quality of dyadic interaction: squality of the relationship and boundary dissolution. Thirteen scales use a seven-point rating system, and one scale, maternal confidence, uses a three-point rating system. Low scores on nine of the scales indicate poor performance (supportive presence, quality of instruction, maternal confidence, child persistence, child enthusiasm, child compliance, child experience, child affect, and quality of relationship). High scores on the five remaining scales indicate child or maternal incompetence (maternal intrusiveness, maternal hostility, child negativity, child avoidance, and boundary dissolution). Studies using the coding scheme have supported the construct, discriminant, criterion, and predictive validity of the measure.^{27,35 – 38}

The first author trained and supervised two research assistants to code the videotaped mother-child interactions using the Egeland et al¹ coding scheme. Training encompassed the review and scoring of 22 pilot tapes until an acceptable level of interrater reliability was achieved. Interrater reliability was reassessed at four intervals throughout the coding process using intraclass correlation coefficients and the kappa statistic, as recommended by the coding manual.¹ Intraclass correlation coefficients for the 13 interval-scaled codes ranged from 0.69 to 0.91 (mean intraclass coefficient = 0.82). Kappa for the single categorical-type item, maternal confidence, was 0.36. Reliability coefficients for the current study were comparable with those reported in the Teaching Task coding manual¹ and previously published manuscripts.³⁵

Past-Year Maternal Drug Use

Maternal Psychosocial Interview—The Psychosocial Interview, administered in a structured format, assesses maternal substance use, family composition, household stability, child-care patterns, support services, and other pertinent psychosocial variables in yearly intervals that are anchored by the index child's birthday. For the present study, information was obtained from the Caregiver Substance Use section, which assesses drug and alcohol use by the mother in the year interval preceding the child's third birthday. In a format similar to the birth interview, the typical dose per day was multiplied by the number of weeks used to obtain an estimate of the total amount of drug use for the prior year, separately for each drug. Maternal cocaine, alcohol, tobacco, and marijuana use in the year preceding the child's third birthday, measured only by self-report, was quantified in this manner. Past-year cocaine use was analyzed as a dichotomous grouping indicator (yes/no), whereas past-year alcohol, tobacco, and marijuana use were analyzed as a continuous composite covariate for each drug, as described above.

Recent Maternal Psychological Distress

Symptom Checklist-90-R—The Symptom Checklist-90-R (SCL-90-R)³⁹ is a 90-item self-report instrument that assesses adult psychological symptoms over the last 7 days. Items are rated using a Likert scale wherein a rating of 0 indicates "not at all distressed" and 4 indicates "extremely distressed" by a particular symptom. For this study, a summary domain from the SCL-90-R, the Global Severity Index (GSI), was used. The GSI assesses overall severity of psychological distress. Reliability coefficient for the overall distress score is 0.60 when the test-retest interval is 1 to 10 weeks.³⁹ In validity studies using the SCL-90-R, adult ratings on the instrument have corresponded with concurrent ratings on the Minnesota Multiphasic Personality Inventory, the Present State Examination, the Center for Epidemiological Studies Depression Scale, and the Beck Depression Inventory.³⁹

Preschool Assessment Procedures

Children were scheduled for the preschool evaluation at approximately 3 months after their third birthday (mean age, 40 mo; range, 36–49 mo). Although the program offered ambulatory pediatric care as an incentive to participation, the preschool developmental visit was scheduled after the routine annual physical examination to minimize subject burden. On the day of the preschool assessment, children completed a standard battery that included the McCarthy Scales of Children's Abilities, as well as other measures of neurodevelopmental functioning, lasting approximately 2.5 hours. Mothers completed a series of demographic and psychosocial questionnaires. On completion of the child and caregiver individual assessments, the mother-child dyad completed a 15-minute, semistructured free-play interaction that was videotaped for later coding.

Protocol for the mother-child semistructured play interaction was as follows. During the videotaped play session an assortment of age-appropriate toys were provided to the dyad to

stimulate spontaneous reciprocal interactions. These toys included Legos, an Ernie doll, two puzzles, three books, a coloring/activity book, a white drawing pad, crayons, a water game, a Barrel of Monkeys, and a flip-up learning center. The toys were presented in a box and placed on a chair between the mother and child. The mother was instructed to play naturally with her child as she typically does at home, and the interaction was terminated at the end of the 15-minute observation period.

Statistical Analysis

Exploratory factor analysis of the 14 Egeland items disclosed a unidimensional latent structure (Factor 1 Eigenvalue = 6.73; cumulative index = 0.79; Factor 2 Eigenvalue = 0.96; cumulative index = 0.90; Factor 3 Eigenvalue = 0.82, cumulative index = 0.99). Internal consistency reliability estimate for the standardized composite Egeland index (mean = 0, SD = 1) was 0.92 with itemmetric analysis showing all alpha at 0.90 or greater when items were removed one by one. Tukey's ladder of powers implemented under STATA software⁴⁰ disclosed no advantage to transformation of the already Gaussian distribution of this composite index. This index was used for the dependent variable in the first series of multiple regression analyses. Individual subscales were then evaluated through multivariate profile analyses.

Multiple regression models were used to estimate the size of the difference in the quality of mother-child interactions under a series of varying model specifications as a function of three primary hypothesized factors: maternal cocaine use during pregnancy (measured dichotomously by combining self-report and toxicology information at birth), maternal cocaine use during the year interval between the index child's second and third birthday (yes/no measured by self-report), and maternal psychological distress in the week preceding the 3-year follow-up visit (GSI score). The stability of the prenatal and past-year cocaine-associated estimates was evaluated with consideration for numerous other suspected determinants of mother-child interaction and evaluated in temporal and conceptual blocks because of sample size limitations. This approach is relatively conservative, evaluating each hypothesized covariate independent of other covariates included in each model. Some suspected determinants of mother-child interaction, however, might also be associated with prenatal cocaine exposure or ongoing maternal cocaine use, indicating more complex interdependent relationships. In the present study, regression modeling was used to evaluate whether the observed cocaine-associated deficit in mother-child interaction should be regarded as independent of other possible determinants or whether results indicate a more complex network of interdependent relationships. As such, interpretation of results is focused on the degree of attenuation of the observed size of the cocaine-associated deficit with statistical adjustment for potentially related covariates. The 95% confidence intervals (CIs) are presented to show the precision of the resulting estimate. In this context, a null result (e.g., $p > .05$) may indicate no effect, but in cases of multicollinearity it may also indicate a more complex pattern of interdependent relationships that cannot be teased apart using single equation multiple regression analyses. Interested readers will find more thorough discussion of these methodological issues in articles and recent textbooks such as Rothman and Greenland.⁴¹

A secondary set of analyses also evaluated the individual subscales of the Egeland et al¹ coding scheme using a multivariate profile analysis⁴² performed with a generalized linear model and generalized estimating equation methods described by Liang and others.^{42,43} This method was first applied to problems of psychological and psychiatric research by Andrade et al⁴⁴ who provide a detailed technical appendix for methodologists. In brief, the generalized linear models with generalized estimating equations (GLM/GEE) multivariate response profile analysis is a multivariate form of multiple regression analysis, with interdependent response variables simultaneously expressed as a function of one or more explanatory characteristics. As a multivariate approach, it provides a simultaneous estimation of the effects of prenatal

cocaine exposure on the Egeland subscales, with consideration for the interrelationships among these outcome measures and with multivariate alpha level correction. Although the commonly used alternative, multivariate analysis of variance, is similar, it requires univariate post hoc follow-up tests. The GLM/GEE approach also uses all available data even when some subjects have missing response values. GLM/GEE estimates are expressed as regression slope coefficients with 95% CI and *p* values presented as an aid to interpretation.

RESULTS

Sample Characteristics

Sample characteristics at birth and 3-year follow-up are presented in Table 1 to Table 3. Table 1 provides an overview of selected maternal and child characteristics at delivery, with a cross-classification by prenatal cocaine exposure status. Table 2 presents descriptive information on maternal prenatal substance use and drug use in the year before the preschool visit. Table 3 summarizes maternal and child demographic characteristics at the time of the preschool assessment, also classified by prenatal substance exposure group. As evident in these tables, the cocaine-exposed group differed from the non-cocaine-exposed group on numerous maternal and child demographic indicators. As noted in the Methods section, the primary research questions were evaluated with inclusion of selected covariates that might also influence mother-child interaction. The stability of the cocaine-related estimates was evaluated and interpreted within the context of these varying models.

Multiple Regression Analyses The models evaluating the hypothesized influence of prenatal cocaine exposure on mother-child interaction are presented in Table 4. The estimated difference (D) from the first regression model, with no statistical adjustment, indicated the Egeland score was approximately one fifth of a SD lower in cocaine-exposed mother-child dyads when compared with the non-cocaine-exposed group (estimated difference = -0.21 , 95% CI = -0.36 , -0.06 , $p = .005$). Model 2 was adjusted for the child's sex and age, with no change in the stability of the estimated difference. The inclusion of terms for child sex and age in subsequent models was based on a guiding conceptual and theoretical model in which we presupposed that mother-child interaction might depend on child sex and age. This modeling approach is conservative in that we can be confident that the estimated cocaine-associated deficit in mother-child interaction is not caused by residual influences of child sex or age, even if the sex- and age-related differences in the Egeland score are not statistically significant by conventional standards (i.e., $p < .05$). As shown in Table 4, Models 3 to 5, the estimates from several additional regression models remained highly consistent (ranging from -0.18 to -0.21 SD) when potential confounding characteristics measured at birth were included in the model.

Table 5 provides evidence of whether mother-child interactions are different for mothers who used cocaine during pregnancy and in the past year, for mothers who used cocaine during pregnancy but not in the past year, and for mothers who abstained from cocaine use both prenatally and in the past year. With no statistical adjustment for prenatal cocaine exposure, the Egeland summary score was 30% of a SD lower when mothers reported cocaine use in the year before the 3-year assessment compared with when mothers did not report past-year cocaine use (estimated difference = -0.03 , 95% CI = -0.48 , -0.11 ; $p = .002$, as shown in Table 5, Model 1).

The reference group for Models 2 to 6 in Table 5 consisted of dyads with no prenatal cocaine exposure (measured by maternal self-report and toxicology at birth) and no self-reported maternal cocaine use during the year before the 3-year assessment ($n = 184$ or 99% of those categorized as non-cocaine-exposed at birth). The estimates presented in Table 5 compare this reference group to dyads with both prenatal cocaine exposure and reported past-year maternal cocaine use ($n = 52$; 33% of those categorized as cocaine-exposed at birth) and to dyads with

prenatal cocaine exposure but no reported past-year maternal cocaine use ($n = 104$; 67% of those categorized as cocaine-exposed at birth).

As indicated in Model 2, Table 5, mother-child dyads with both prenatal and past-year maternal cocaine use had Egeland summary scores an estimated 36% of a SD lower than the reference group of dyads (95% CI = $-0.56, -0.16$; $p = .001$). The influence of prenatal cocaine exposure in the absence of past-year maternal cocaine use on mother-child interaction was not remarkable when compared with the reference dyads ($D = -0.14$; 95% CI = $-0.31, 0.03$; $p = .108$). Gauged in relation to their statistical precision, these two estimates (-0.36 vs -0.14) were different from one another ($p = .046$).

Model 3 evaluated the hypothesized relationship between maternal psychological distress in the week preceding the 3-year visit and mother-child interaction, independent of maternal cocaine use. Results indicated no additional contribution of maternal psychological distress independent maternal maternal drug use variables ($p = .233$). When considered without statistical control for maternal cocaine use, results were similar ($p = .145$; data not shown in table). Also depicted in Model 3, Table 5, the estimates for past-year maternal cocaine use alone and in combination with prenatal cocaine exposure remained stable with statistical control for maternal Global Severity Index (GSI).

Model 4 of Table 5 indicates that the size of the cocaine-associated deficit is not independent of past-year maternal use of alcohol, tobacco, and marijuana. With statistical adjustment for past-year use of these drugs, there was an appreciable attenuation of the estimated size of the mother-child interaction deficit, not only for children whose mothers used cocaine during pregnancy without cocaine use in the past year ($D = -0.05$, 95% CI = $-0.23, 0.14$, $p = .621$) but also for those with both prenatal cocaine exposure and past-year maternal cocaine use ($D = -0.24$, 95% CI = $-0.49, 0.01$, $p = .054$). This attenuation, however, must be evaluated with consideration for the overlap among the past-year substance use variables, particularly tobacco use, with prenatal cocaine exposure (Table 3). These regression models make statistical adjustments for alcohol, tobacco, and marijuana use as if they were strictly independent of maternal cocaine use, when in fact the use of alcohol, tobacco, marijuana, and cocaine are interdependent (e.g., a cocaine binge might prompt self-medication in the form of drinking alcohol or smoking marijuana).

Models 5 and 6, presented in Table 5, show relative stability of the cocaine-associated estimates when considering a number of covariates assessed at the 3-year follow-up visit. For example, with statistical adjustment for past-year marital status, employment, and educational attainment (Model 5), the Egeland score for children whose mothers used cocaine both during pregnancy and in the year before the 3-year visit was an estimated 30% of a SD lower than scores for nonexposed children ($D = -0.30$, 95% CI = $-0.51, -0.09$; $p = .006$). With the same statistical adjustment, the size of the estimated mother-child interaction deficit associated with prenatal cocaine exposure was somewhat smaller when there was no past-year cocaine use ($D = -0.11$; 95% CI = $-0.27, 0.06$; $p = .198$). Similarly, when the model was adjusted for child cognitive status, child blood lead levels, number of caregiver changes, daycare attendance, and special developmental services, the estimate for prenatal cocaine exposure in combination with maternal past-year cocaine use remained stable ($D = -0.34$; 95% CI = $-0.55, -0.12$; $p = .003$); the estimate for children with only prenatal cocaine exposure reached conventional levels of statistical significance when these covariate adjustments were included ($D = -0.18$; 95% CI = $-0.35, -0.01$; $p = .044$).

Multivariate Profile Analysis

Table 6 presents results from the multivariate analysis of Egeland subscales, based on the GLM/GEE profile analysis method (also depicted graphically in Fig. 1). Based on conventional

standards of statistical significance ($p < .05$), the evidence of cocaine-associated deficits (impairments) in mother-child interactions is strongest with respect to maternal intrusiveness, hostility, quality of instruction, confidence, and child persistence (Table 6). With statistical adjustment for past-year maternal cocaine use and other covariates, the strength of the evidence was reduced with respect to child persistence ($p = .16$) but not with respect to maternal intrusiveness ($p < .001$), hostility ($p < .001$), quality of instruction ($p < .001$), and maternal confidence ($p = .026$) (data not shown in table). Covariates included in this adjustment were child sex and age, prenatal exposure to marijuana, alcohol, and tobacco; past-year maternal use of cocaine, alcohol, marijuana, and tobacco; maternal educational, marital, and employment status at the time of the 3-year assessment; and the child's receipt of day care or special developmental services in the preceding year.

DISCUSSION

Findings from the present study indicate that prenatal cocaine exposure, without adjustment for past-year maternal cocaine use, is associated with poorer summary ratings of mother-child interactions measured at 3 years of age. The strength of the estimated difference in mother-child interactions between cocaine-exposed and non-cocaine-exposed dyads remained stable with adjustment for numerous other characteristics measured at birth, including the child's age and sex; prenatal exposure to alcohol, tobacco, and marijuana; maternal age, education, marital status, and employment; and the mother's primigravida status and prenatal care.

Maternal cocaine use in the past year was also associated with poorer ratings of mother-child interaction measured at 3 years of age. When considered together with prenatal cocaine use, the association between prenatal cocaine exposure and mother-child interaction was not independent of the relationship between past-year maternal cocaine use and mother-child interaction. The strength of the associated deficit in mother-child interactions was greatest for children with both prenatal cocaine exposure and ongoing maternal cocaine use when compared with children whose mothers did not use cocaine during pregnancy or at the 3-year follow-up visit (all but two of the original non-cocaine-exposed group). For children with prenatal cocaine exposure whose mothers did not report past-year cocaine use, mother-child interactions did not appear to be different from children with no known cocaine exposure under most model specifications. These findings may indicate a potential cumulative influence for children with a predisposed risk as a result of prenatal cocaine exposure whose mothers continue to use cocaine and other drugs, although such conclusions are beyond the scope of the study.

Although the prenatal and past-year cocaine-associated differences were prominent in the current study, there was no noteworthy association between levels of maternal psychological distress in the week preceding the 3-year assessment and parent-child interactions. In the present sample, levels of reported maternal psychological distress were very similar in the cocaine-exposed and non-cocaine-exposed groups.

Multivariate analyses provided additional information regarding the specific subscales of the Egeland parenting construct in relation to maternal cocaine use during pregnancy. Specifically, the most pronounced differences between cocaine-exposed and non-cocaine-exposed dyads were in relation to ratings of maternal characteristics such as hostility, intrusiveness, quality of instruction, and confidence. Aside from an observed difference between cocaine-exposed and non-cocaine-exposed children in task persistence during the interaction ($p < .05$), no additional differences were noted on other child characteristics such as child negativity, child compliance, affect toward mother, and avoidance.

Overall, findings indicate that the process of mutual regulation that occurs in healthy mother-child interactions may be somewhat disrupted in cocaine-exposed children, particularly when maternal cocaine use continues during the early childhood period. This risk may not be as significant in the absence of ongoing maternal drug use; however, it is difficult to tease apart the relative contribution of each of these factors given the high degree of concordance in the current sample between those mothers with known prenatal cocaine use and those who admitted continued use. It was also not possible to evaluate the association between past-year maternal cocaine use in the absence of prenatal cocaine exposure, because only two mothers from the non-cocaine-exposed group reported past-year consumption of cocaine.

The observed decrement in mother-child interactions was not statistically independent of mothers' past-year alcohol, tobacco, and marijuana use. In the current sample, maternal cocaine use frequently occurred in conjunction with tobacco, alcohol, and marijuana use, making it difficult to separate out a cocaine-specific influence. Prenatal cocaine exposure is believed to have a teratological influence on infant neurobehavioral functioning, impairing the infant's ability to self-regulate during dyadic interactions. Prenatal cocaine exposure has been associated with impaired behavioral and emotional regulation during the infancy and early preschool years.⁴⁵ Conversely, ongoing maternal cocaine use, particularly in combination with other drugs, may influence the quality of caregiving behavior, negatively impacting the nature of the dyadic interchange. Here, the issue speaks to the degree that caregiving behavior might be impaired by maternal drug use, rather than delineation of a drug-specific effect. Results from the current investigation indicate a potential combinatorial influence, with prenatal cocaine exposure placing the child at increased risk for regulatory difficulties that may be further exacerbated by ongoing maternal drug use and impaired maternal caregiving behavior.

The current study is one of the few investigations to examine the long-term influence of prenatal cocaine exposure on parent-child interaction beyond the infancy and toddler period. Also, in contrast with much of the existing literature on parenting and maternal substance use, this project followed a cohort of children who were enrolled prospectively at birth, with prenatal cocaine use measured by maternal self-report immediately postpartum, and infant and maternal biological markers taken at the time of delivery. Enrollment of the study sample was restricted to African-American mothers with full-term infants. Furthermore, mother-child interactions were assessed directly using standardized mother-child observations as opposed to indirectly through maternal self-report questionnaires or reports of child abuse and neglect. The study also used a modified version of an observational coding scheme that has been extensively tested with low-income and at-risk samples.^{21–23} Raters were blind to the prenatal drug status of the mother-child dyads, and maternal reports of past-year substance use were collected by interviewers who did not participate in the videotaped mother-child interaction protocol.

Nonetheless, study results should be interpreted in light of certain limitations. The study relied on maternal self-report for the assessment of past-year substance use. Despite protection from a federal Department of Health and Human Services (DHHS) Certificate of Confidentiality, the possible underreporting of past-year drug and alcohol use may have reduced the strength of the study's estimate of the association between past-year cocaine use and mother-child interactions. Another possible limitation of the study is the restriction of the sample to full-term infants, as this may have attenuated the relationship between prenatal cocaine exposure and mother-child interactions. The present study included only children remaining in the care of their biological mothers at the time of the preschool assessment, which may have biased findings by eliminating a subset of children who were placed in alternative care, perhaps because of a more severe cocaine-use problem by the mother. Finally, the study population was not large or heterogeneous enough to allow for the examination of past-year cocaine use by mothers in the absence of prenatal cocaine use.

The current study's finding of more problematic mother-child interactions in children exposed to cocaine prenatally has been indicated in prior studies, although with somewhat less control over confounding influences.^{5,6} In the present study, cocaine-using mothers had composite parent-child interaction scores that were lower than noncocaine-using mothers, and when interaction ratings were examined independently, cocaine-using mothers showed higher levels of negative parenting behaviors (e.g., hostility and intrusiveness) and lower levels of positive parenting behaviors (e.g., instruction and confidence). Other authors have reported that prenatal cocaine-using mothers are less available, less resourceful, less reinforcing, and more likely to use physical discipline than are noncocaine-using mothers.^{5,6} Taken together, these findings provide support for the conclusion that mothers who use cocaine prenatally are at risk for a variety of adverse parenting and parent-child relationship outcomes.

A more difficult finding to explain is the lack of significant differences between the cocaine-exposed and non-cocaine-exposed children on the Egeland et al¹ subscales, aside from a difference in child persistence. Available evidence indicates that cocaine-exposed children experience greater difficulty in emotional and behavioral regulation than do non-cocaine-exposed children,^{46,47} and so it was anticipated that cocaine-exposed preschoolers would have more problems responding to and interacting with their mothers than the nonexposed children. Perhaps the nature of the interaction, a semistructured play session with a set of novel toys, was too intriguing to create a strong or negative demand on the child. Furthermore, although the children appeared to have experienced the interaction as one calling for playful exchanges, many of the study mothers interpreted the play interaction as a situation calling for structure, discipline, and displays of child achievement, making the interaction more stressful from their point of view.

In conclusion, both prenatal and past-year cocaine use were associated with parent-child interaction deficits in the early preschool period, with mother-child interaction being most affected when prenatal exposure was combined with ongoing maternal cocaine use. In addition to experiencing the putative effects of prenatal cocaine exposure, some of these children may face social adaptation challenges in their interactions with their mothers who used cocaine during and after pregnancy. Whether these differences will produce lasting effects of clinical significance remains to be seen in future longitudinal research with samples large enough to capture a broad range of variation. These results, if confirmed, have implications for clinicians working with high-risk children and families. In particular, health care providers should assess whether pediatric patients have been exposed to cocaine or other drugs during or after pregnancy and should take maternal drug use into account when evaluating mother-child interactions and child behavior problems. Future research is needed to better understand the mechanisms underlying these cocaine-associated deficits and whether specific interventions might be able to improve or ameliorate the types of deficits observed.

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FIGURE 1.

Maternal-child interaction subscales in prenatally cocaine-exposed and non-cocaine-exposed dyads. To simplify interpretation of the multivariate model, the Egeland subscales were rescaled with 0 as the lowest value (i.e., a value of 1 was subtracted from each rating). $*p < .05$.

Table 1

Maternal and Infant Demographic Sample Description at Birth (n = 343)

	Non-Cocaine-Exposed (n = 186)	Cocaine-Exposed (n = 157)
Maternal characteristics		
Maternal age **	23.8 (5.6)	28.6 (4.8)
Education (yr)	11.2 (1.4)	11.3 (1.3)
Unemployed **	84%	93%
Never married	89%	90%
Prenatal care (≤ 3 visits) *	15%	26%
Primigravida **	24%	8%
Infant characteristics		
Male	51%	49%
Birth weight (g) **	3300 (490)	2.999 (476)
Birth length (cm) **	50.8 (2.3)	49.1 (2.5)
Birth head circumference (cm) **	33.8 (1.4)	33.1 (1.5)
Gestational age (wk) *	39.7 (1.4)	39.3 (1.4)

Numbers represent means (SD) or percentages where indicated.

*
p < .05**
p < .01.

Table 2

Maternal Self-Reported Alcohol, Tobacco, Marijuana, and Cocaine Use (n = 343)

	Non-Cocaine-Exposed (n = 186)		Cocaine-Exposed (n = 157)	
	Median (Min, Max) ^a	% (n)	Median (Min, Max) ^a	% (n)
Prenatal substance use				
Cocaine/crack (no. lines/rocks)	–	–	86 (1, 19320)	64(100)
Alcohol (no. drinks) ^c	54(2, 1680)	28 (53)	74 (1, 3823)	63 (99)
Marijuana (no. joints) ^c	37 (1, 807)	12 (22)	28 (1, 798)	42 (66)
Tobacco (no. cigarettes) ^{b,c}	700 (3, 5880)	16 (29)	1974(2, 8820)	71 (112)
Postnatal substance use				
Cocaine/crack (no. lines/rocks) ^c	78 (52, 104)	1.1 (2)	56 (1, 728)	33 (52)
Alcohol (no. drinks) ^b	44 (1, 416)	40 (74)	104 (1, 728)	45 (70)
Marijuana (no. joints) ^c	104 (1, 364)	11 (21)	94 (8, 364)	26 (40)
Tobacco (no. cigarettes) ^{b,c}	364 (104, 364)	16 (30)	364 (52, 364)	66 (103)

^aMedian values based only on mothers reporting usage, calculated using total exposure composites: number of weeks used × usual number of days per week × usual dose per day.

^b $p < .05$, comparison of median alcohol, tobacco, and marijuana use (columns 1 and 3).

^c $p < .05$, comparison of proportion of mothers reporting alcohol, tobacco, and marijuana use (columns 2 and 4).

Table 3
 Caregiver and Child Demographic Sample Description at 3-Year Visit (n = 343)

	Non-Cocaine- Exposed (n = 186)	Cocaine- Exposed (n = 157)
Maternal characteristics		
Mother's age **	27.2 (5.6)	32.0 (4.8)
Education (yr)	11.3 (1.3)	11.1 (1.4)
Unemployment **	64%	81%
Never married	76%	75%
Past-year cocaine use **	1%	33%
Past-year alcohol use	40%	45%
Past-year tobacco use **	16%	66%
Past-year marijuana use **	11%	26%
Global Severity Index (GSI)	55.8 (11.0)	55.8 (11.8)
Child characteristics		
Child Age (mo) *	40.5 (3.6)	39.7 (3.0)
Male	51%	49%
Intellectual Functioning (GCI)	82.4(13.0)	81.8 (10.7)
Child in day care	34%	43%
Child receiving special services	1%	3%
Children with one or more caregiver changes in past 3 years **	5%	15%
Number of caregiver changes in past 3 yr (limited to children with one or more changes)	1.4(0.52)	1.3 (0.63)

GCI, General Cognitive Index.

Numbers represent means (SD) or percentages where indicated.

*
 $p < .05$

**
 $p < .01$.

Table 4

Multiple Regression Models to Estimate the Egeland Summary Score Difference Between Prenatally Cocaine-Exposed and Non-Cocaine-Exposed Children (n = 343)

	Estimated Difference ^a	95% CI	p Value
Model 1			
Prenatal cocaine exposure (no other covariates)	−0.21	−0.36, −0.07	.005
Model 2			
Prenatal cocaine exposure, child sex, and test age	−0.20	−0.35, −0.06	.007
Model 3			
Subsumes Model 2, plus terms for level of prenatal exposure to alcohol, marijuana, and tobacco based on maternal self-report	−0.18	−0.34, −0.02	.028
Model 4			
Subsumes Model 2, plus terms for maternal age, years education, marital status, and employment	−0.18	−0.34, −0.01	.042
Model 5			
Subsumes Model 2, plus terms for primigravida status and prenatal care visits	−0.21	−0.37, −0.06	.008

CI, confidence interval.

^aEstimated difference associated with prenatal cocaine exposure, controlling for birth covariates. Difference estimates expressed in SD units for the Egeland Summary Score.

Table 5

Multiple Regression Models to Estimate the Egeland Summary Score Difference for Maternal Prenatal and Past-Year Cocaine Use at the 3-Year Follow-Up Visit

	Estimated Difference ^a	95% CI	p Value
Model 1 (n = 342)			
Maternal past-year cocaine use	−0.30	−0.48, −0.11	.002
Model 2 (n = 342)			
Prenatal cocaine exposure with maternal past-year cocaine use	−0.36	−0.56, −0.16	.001
Prenatal cocaine exposure without maternal past-year cocaine use	−0.14	−0.31, 0.03	.108
Model 3 (n = 340)			
Maternal psychological symptoms (GSI score)	−0.04	−0.12, 0.03	.233
Prenatal cocaine exposure with maternal past-year cocaine use	−0.35	−0.55, −0.14	.001
Prenatal cocaine exposure without maternal past-year cocaine use	−0.14	−0.31, 0.03	.096
Model 4 (n = 334) (adjusted for maternal past year alcohol, tobacco, marijuana use)			
Prenatal cocaine exposure with maternal past-year cocaine use	−0.24	−0.49, 0.01	.054
Prenatal cocaine exposure without maternal past-year cocaine use	−0.05	−0.23, 0.14	.621
Model 5 (n = 340) (adjusted for current education, employment, and marital status)			
Prenatal cocaine exposure with maternal past-year cocaine use	−0.30	−0.51, −0.09	.006
Prenatal cocaine exposure without maternal past-year cocaine use	−0.11	−0.27, 0.06	.198
Model 6 (n = 323) (adjusted for child cognitive status [GCI], blood lead levels, number of caregiver changes, daycare attendance, and special developmental services)			
Prenatal cocaine exposure with maternal past-year cocaine use	−0.34	−0.55, −0.12	.003
Prenatal cocaine exposure without maternal past-year cocaine use	−0.18	−0.35, −0.01	.044

^aEstimated difference associated with prenatal cocaine exposure, controlling for birth covariates. Difference estimates expressed in SD units for the Egeland Summary Score.

Table 6

Multivariate Contrasts of Egeland Interaction Subscales Between Prenatally Cocaine-Exposed and Non-Cocaine-Exposed Groups (n = 343)

Egeland Subscales	Cocaine-Exposed (n = 157)		Non-Cocaine-Exposed (n = 186)		p Value
	Estimate	95% CI	Estimate	95% CI	
Maternal Intrusiveness	2.30	2.04, 2.57	1.52	1.31, 1.73	<.001
Maternal Hostility	2.00	1.74, 2.26	1.30	1.07, 1.52	<.001
Quality of Instruction	2.22	1.98, 2.45	2.73	2.50, 2.97	.002
Maternal Confidence	1.71	1.40, 2.03	2.37	2.04, 2.70	.004
Maternal Support	2.50	2.26, 2.74	2.85	2.59, 3.11	.051
Child Persistence	2.71	2.50, 2.92	3.06	2.87, 3.26	.014
Child Enthusiasm	2.73	2.51, 2.95	2.99	2.79, 3.20	.080
Child Negativity	1.83	1.58, 2.07	1.59	1.37, 1.81	.161
Child Compliance	3.30	3.07, 3.53	3.47	3.26, 3.69	.275
Child Experience of Session	2.82	2.60, 3.05	3.12	2.90, 3.45	.061
Child Affect to Mother	2.85	2.63, 3.08	3.04	2.83, 3.27	.221
Child Avoidance	2.15	1.87, 2.44	2.24	1.97, 2.51	.655
Quality of the Relationship	2.31	2.07, 2.54	2.55	2.31, 2.78	.154
Boundary Dissolution	2.17	1.93, 2.42	2.10	1.89, 2.32	.672

CI, confidence interval.

To simplify interpretation of results from this multivariate model, the Egeland subscale scores were rescaled with 0 as the lowest value (i.e., a value of 1 was subtracted from each rating).