Expressive and Receptive Language Functioning in Preschool Children With Prenatal Cocaine Exposure

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Objective  To estimate the relationship between severity of prenatal cocaine exposure and expressive and receptive language skills in full-term, African American children at age 3 years. Methods  Language was assessed at age 3 using the Clinical Evaluation of Language Fundamentals–Preschool (CELF-P). The sample included 424 children (226 cocaine exposed, 198 non–cocaine exposed) who received preschool language assessments at age 3, drawn from a cohort of 476 children enrolled prospectively at birth. Results  Structural equation modeling was used to regress expressive and receptive language as intercorrelated response variables on level of prenatal cocaine exposure, measured by a latent construct including maternal self-report of cocaine use and maternal/infant urine toxicology assays and infant meconium. Results indicated a .168 SD decrease in expressive language functioning for every unit increase in exposure level (95% CI = –.320, –.015; p = .031) after consideration for fetal growth and gestational age as correlated response variables. Receptive language was more modestly related to prenatal cocaine exposure and was not statistically significant. Results for expressive language remained stable with inclusion of the McCarthy general cognitive index as a response variable (expressive language $\beta = –.173$, 95% CI = –.330, –.016; p = .031), and with adjustment for maternal age and prenatal exposures to alcohol, tobacco, and marijuana (expressive language $\beta = –.175$, 95% CI = –.347, –.003; p = .046). Additional child and caregiver environmental variables assessed at age 3 were also evaluated in varying statistical models with similar results. Conclusion  The evidence from this study supports a gradient relationship between increased level of prenatal cocaine exposure and decreased expressive language functioning in preschool-aged cocaine-exposed children.

Key words  prenatal cocaine exposure; language functioning; preschool children; CELF-P.

The clinical impact of prenatal cocaine exposure on long-term child health and development remains a compelling topic of interest for health care providers. Cocaine readily crosses the placenta and has been associated with alterations in the developing monoaminergic neurotransmitter systems, with implications for both structural and functional aspects of fetal brain development (Mayes, 1999). Cocaine-associated maternal hypertension, decreased uterine blood flow, fetal vasoconstriction and hypoxemia (Moore, Sorg, Miller, Key, & Resnik, 1986; Volpe, 1992), and nutritional deficiencies may also indirectly influence fetal neurodevelopment (Frank et al., 1990). Research from the infancy and toddler periods suggests that prenatal cocaine exposure may lead to subtle neurobehavioral
impairments, increasing long-term risk for learning and social/behavioral difficulties in children (Carmichael Olson & Toth, 1999; Lester, LaGasse, & Seifer, 1998).

Characterizing the early language development of cocaine-exposed children is of particular concern, due to the complex interplay between language acquisition, cognitive development, and learning, as well as the potential implications for long-term social adaptation and academic success. While much has been written regarding the potential teratogenic impact of cocaine on developing monoaminergic neurotransmitter systems in the fetus, the specificity of how cocaine might influence evolving neurological substrates and biochemical processes in relation to language development is not well understood, and would likely be greatly influenced by factors such as timing and degree of exposure as well as individual differences in maternal and placental metabolism. It has been hypothesized, however, that early language development may be affected through three potentially interactive pathways, including the subtle disregulation of attentional systems within the brain that impedes processing of available linguistic information, disruptions in parent-child linguistic interactions due to parental cocaine and other drug use, and impaired learning due to the unstable and potentially impoverished caregiving environments associated with caregiver drug use and poverty (Malakoff, Mayes, Schottenfeld, & Howell, 1999).

The extant published literature describing language development in cocaine-exposed children largely emanates from smaller studies, frequently characterized by limitations in sampling techniques and exposure status classification. Studies have reported language deficits on various standardized tests of global language functioning (Johnson, Seikel, Madison, Foose, & Rinard, 1997), and expressive and receptive language abilities in cocaine-exposed children during the preschool period (Bender et al., 1995; Koren et al., 1998; Nulman et al., 2001). More recently, findings from larger well-controlled prospective studies have also put forth evidence suggesting that cocaine-exposed children may be at increased risk for language difficulties. For example, Singer et al. (2001) documented lower total language scores and poorer auditory comprehension in more heavily cocaine-exposed infants, and Delaney-Black et al. (2000) reported an increased likelihood of cocaine-exposed children being categorized as low language functioning at age 6, although they did not find mean differences on standardized language measures or expressive language samples. Several other reports, however, including a relatively large prospective study conducted by Hurt, Malmud, Betancourt, Brodsky, and Giannetta (1997), have not documented differences in language abilities on standardized assessments (Espy, Kaufmann, & Glisky, 1999; Phelps & Cottone, 1999).

While an emerging pattern of cocaine-related language effects is suggested by many early studies, methodological limitations including small sample sizes, nonprospective study designs, and failure to statistically control for important confounding influences have made it difficult to ascertain a cocaine-specific effect on language development. The Miami Prenatal Cocaine Study (Miami PCS) is a longitudinal investigation of the effects of prenatal cocaine exposure on child development in a large (N = 476), well-retained cohort, enrolled prospectively at birth with verification of substance-exposure status using biological markers in addition to maternal self-report. Previous published reports from this cohort have shown cocaine-associated decrements in the early language development of cocaine-exposed children (Morrow et al., 2003). Specifically, global language acquisition was measured longitudinally at 4, 8, 12, 18, and 24 months using items drawn from the Bayley Scales of Infant Development (Bayley, 1993) and linked to language skills assessed at age 3 years using the Clinical Evaluation of Language Fundamentals–Preschool (CELF-P) (Wiig, Secord, & Semel, 1992). Longitudinal analyses indicated that prenatally cocaine-exposed children had lower overall language skills than non-cocaine-exposed children, although the mean difference between the groups was modest (15% of a standard deviation). These findings were extended longitudinally through age 7, again using measures of global language functioning with similar results (the estimated mean difference between the cocaine-exposed and non-cocaine-exposed children was 17% of a standard deviation) (Bandstra et al., 2002). Taken together, these reports support a stable but modest cocaine-specific effect on summary indicators of language functioning in cocaine-exposed children when compared with non-cocaine-exposed children during early childhood, but they do not address the more specific processing modalities of language development.

In addition, few studies have attempted to document a relationship between the severity of prenatal cocaine exposure and the degree of language impairment, an important step in determining the teratogenic impact of cocaine use during pregnancy on child developmental functioning. A smaller study relying on maternal self-report to document severity of exposure found no decrement in language functioning (Hurt et al., 1997). Singer and colleagues (2001), however, found lower auditory comprehension and total language scores in children identified as having higher prenatal cocaine exposure levels. Longitudinal findings from the current cohort
also support a gradient relationship between severity of exposure and decrements in global language functioning. Bandstra et al. showed a significant relationship between severity of prenatal cocaine exposure and summary language scores over the span of 3 to 7 years using longitudinal latent growth curve modeling (Bandstra, Vogel, Morrow, Xue, & Anthony, 2004). These analytic models, however, focused on a single outcome measure of global language functioning without consideration for the subcomponents of language processing. The current report augments these findings and other published works by evaluating components of receptive and expressive language processing at age 3 years in relation to the severity of prenatal cocaine exposure in a large, well-retained cohort of children. A gradient relationship between exposure level and expressive and receptive language functioning was hypothesized. Structural equation modeling (SEM) utilizing a latent construct for prenatal cocaine exposure that included all available self-report and toxicology information was used to evaluate the hypothesized relationships, including statistical adjustment for other prenatal drug exposures and potential confounding influences.

Methods
Study Recruitment and Participants
The study sample of 476 infants (253 cocaine exposed and 223 non–cocaine exposed) was recruited at birth between November 1990 and July 1993 as part of a larger epidemiological study. Recruitment procedures have been extensively detailed in an earlier report (Bandstra et al., 2001). The study was approved by the institutional review board and conducted under a federal Department of Health and Human Services certificate of confidentiality. Informed consent was obtained from all participants. Experienced research staff performed a standardized research interview and collected the biological specimens during the postpartum period. Trained research personnel, blinded to exposure status, performed the Ballard gestational age assessment (Ballard, Novak, & Driver, 1979) within 36 hours of delivery and obtained occipital–frontal head circumference and recumbent crown–heel birth length. Pertinent medical and demographic data were collected from the hospital record at birth.

The study sample was homogeneous with regard to full-term gestational age (≥237 completed weeks), low socioeconomic status, inner-city residence, and African American race/ethnicity. Table 1 shows maternal and infant characteristics at delivery. Exclusion criteria included maternal HIV/AIDS; prenatal exposure to opiates, methadone, amphetamines, barbiturates, benzodiazepines, or phencyclidine; major congenital malformation; chromosomal aberration; or disseminated congenital infection. The sample included 253 cocaine-exposed infants (with varying exposures to 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. Prenatal cocaine exposure was determined by maternal self-report and positive assay on one or more biological markers, including maternal urine, infant urine, and meconium. Alcohol and tobacco exposures were determined by self-report, and marijuana exposure was indicated by self-report and/or a positive toxicology screen. Drug-free mothers had negative self-report drug histories during and for 3 months preceding pregnancy, negative lifetime histories for cocaine use, and negative results on all available toxicology assays.

From the original cohort of 476 infants, 424 (198 non–cocaine exposed, 226 cocaine exposed) completed a valid preschool assessment and were included in the present report. A total of 439 children (92% of the original cohort) returned for the 3-year preschool assessment; of these, 15 children did not complete valid language assessments and were not included in analyses (7 due to refusal/inhibited behavior and 8 due to extreme cognitive delays). There were no group differences in the proportion of children unable to complete the exams.
Drug Exposure Measures at Birth

Maternal Interview

Mothers were interviewed at birth regarding their drug use during pregnancy. Standardized drug-use questions were asked by trimester and included number of weeks used, most days per week, fewest days per week, usual number of days per week, and usual dose per day. Dosage was measured in number of cigarettes smoked, number of marijuana joints smoked, number of drinks of beer, wine, or hard liquor, and number of cocaine lines/rocks, recorded in increments of usual daily dose, usual number of days per week, and number of weeks used. Standard definitions were used for determining 1-drink units for each type of alcohol (beer, 12 oz; wine, 5 oz; and liquor, 1.5 oz). Pregnancy exposure composites were calculated for each drug by multiplying the number of weeks used by the usual days per week and the usual dose per day. Median usage levels for each drug are presented in Table I.

Biological Markers (Urine and Meconium)

Screening of maternal and infant urine and infant meconium for cocaine metabolite (benzoylecgonine) was performed by EMIT® (Syva DAU), 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 (GC/MS) (Mulé & Casella, 1988). Urine specimens were also assayed by EMIT® for marijuana, opiates, amphetamines, barbiturates, benzodiazepines, and phencyclidine. Meconium specimens were assayed by EMIT® for marijuana and opiates. In the original cohort, 100% had at least one biological marker, 96% had at least two biological markers, and 68% had all three biological markers.

Three-Year/Preschool Follow-Up Measures

Child Assessments. The CELF-P (Wiig, Secord, & Semel, 1992) was administered at the 3-year preschool research visit. The CELF-P is an individually administered test of expressive and receptive language ability for children ages 3 through 6 years. The test yields standard scores for receptive subtests (linguistic concepts, sentence structure, and basic concepts) and expressive subtests (recalling sentences in context, formulating labels, and word structure) and composite scores for total language, receptive language, and expressive language. The CELF-P was standardized on 800 preschoolers, representative of the U.S. population with regard to gender, race/ethnicity, parent education, and geographical region. Internal consistency estimates for composite scores ranged from .73 to .96 across age groups, with test-retest coefficients ranging from .87 to .97. The CELF-P correlates with the preschool language scale (third edition) (.90) and the

Table I. Maternal and Infant Characteristics at Birth Enrollment (n = 424)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Non-cocaine-exposed (n = 198)</th>
<th>Cocaine-exposed (n = 226)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Maternal Characteristics at Birth</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal age (years)*</td>
<td>23.8 (5.6)</td>
<td>28.8 (4.8)</td>
</tr>
<tr>
<td>Prenatal care ≥ 4 visits (%)*</td>
<td>84.9</td>
<td>69.0</td>
</tr>
<tr>
<td>Unemployed (%)*</td>
<td>83.3</td>
<td>94.3</td>
</tr>
<tr>
<td>Education (years)</td>
<td>11.3 (1.4)</td>
<td>11.1 (1.4)</td>
</tr>
<tr>
<td><strong>Infant Characteristics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Birth weight (gm)*</td>
<td>3306.7 (492)</td>
<td>2943.4 (470)</td>
</tr>
<tr>
<td>Birth length (cm)*</td>
<td>50.8 (2.3)</td>
<td>48.8 (2.5)</td>
</tr>
<tr>
<td>Birth head circumference (cm)*</td>
<td>33.8 (1.4)</td>
<td>33.0 (1.5)</td>
</tr>
<tr>
<td>Gestational age (wks)*</td>
<td>39.7 (1.4)</td>
<td>39.3 (1.4)</td>
</tr>
<tr>
<td>Male (%)</td>
<td>49.5</td>
<td>49.1</td>
</tr>
<tr>
<td><strong>Self-Reported Maternal Drug Use During Pregnancy</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-reported median reported use</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol (# drinks)*</td>
<td>54 (2,1680)</td>
<td>30.8 (61)</td>
</tr>
<tr>
<td>Tobacco (# cigarettes)*</td>
<td>700 (1,5880)</td>
<td>16.7 (33)</td>
</tr>
<tr>
<td>Marijuana (# joints)*</td>
<td>37 (1,807)</td>
<td>11.1 (22)</td>
</tr>
<tr>
<td>Cocaine/crack (# lines/rocks)</td>
<td>127 (1,19320)</td>
<td>69.0 (156)</td>
</tr>
</tbody>
</table>

Note: Maternal and infant characteristics are expressed as mean (standard deviation) or percentages where indicated. Maternal substance use is expressed as median (min/max) due to skewed distributions.

*p < .01, between group comparisons of maternal and infant characteristics.

*p < .001, between group comparisons of median self-reported maternal drug use (columns 1 and 3).

Median values based only on mothers reporting use, calculated using total exposure composites: (number of weeks used) × (usual number of days per week) × (usual dose per day)
verbal IQ scale of the Wechsler Preschool and Primary Scale of Intelligence–Revised (WPPSI-R) (.72). The McCarthy Scales of Children’s Abilities (McCarthy, 1972), a cognitive assessment for children ages 2 through 8 years with established reliability and validity, was also administered at age 3 and was included in the present report as a covariate. The McCarthy was selected for the 3-year assessment over other instruments available during the early to mid-1990s, when the data were collected, due to its shorter administration time, child-friendly items for assessing very young children, and inclusion of a motor assessment scale. Studies indicate that the McCarthy yields similar IQ scores when compared with the WPPSI-R, with full-scale validity correlations at .68 (Sattler, 2001).

**Child Hearing.** Hearing was assessed using play audiometry techniques at age 3. For children who were unable to complete the play audiometry task, visual reinforcement audiometry in the sound field was used. All testing and interpretations were performed at the University of Miami Mailman Center for Child Development by a licensed, certified pediatric audiologist. For the current analyses, behavioral audiometry results were coded as normal in at least one ear or bilaterally abnormal. Minimum response levels of 30 dB were considered abnormal.

**Blood Lead Levels.** Screening lead levels were performed at age 3 by capillary sample and processed at the State of Florida Department of Health Laboratory. Abnormal capillary lead levels (i.e., ≥10 µg/dl) were confirmed by repeat specimen obtained by venipuncture.

**Caregiver Psychosocial Interview.** A structured psychosocial interview covering family and demographic information was conducted with the mother/primary caregiver of each child at the 3-year visit. The primary caregiver was defined as any family member or custodial guardian responsible for the physical, emotional, and financial well-being of the child. Biological mothers residing with and parenting the child were always prioritized for interview purposes as the primary caregiver.

**Statistical Analyses**

Initial data review procedures included visual inspection of frequency distributions and longitudinal plots for individual subjects. SEM as generalized by Muthén for categorical variables was then used to construct a series of models to estimate the influence of prenatal cocaine exposure on expressive and receptive language. Mplus software (Muthén & Muthén, Los Angeles, CA) was used for all analyses. Effect estimates, 95% confidence intervals, and p-values are presented for interpretation of results. Level of prenatal cocaine exposure was measured by a single summary latent construct comprising information from two latent constructs: (1) intercorrelated bioassay results (cocaine-positive [yes/no] of infant urine, infant meconium, and maternal urine; and log-transformed levels of cocaine metabolite benzoylecgonine determined by GC/MS assays) and (2) intercorrelated composites of self-reported amount of cocaine use for each trimester, categorized in four levels (0 = no self-reported cocaine/crack use, 1 = 1–24 uses, 2 = 25–180 uses, 3 = 180+ uses2). This latent construct approach allowed for bioassay results to characterize cocaine exposure levels for all infants, including known cocaine-positive infants whose mothers denied cocaine use.

The SEM approach was then used to model latent constructs for the CELF-P expressive subscale standard scores (recalling sentences in context, formulating labels, and word structure) and receptive subscale standard scores (linguistic concepts, sentence structure, and basic concepts) as separate intercorrelated constructs, allowing for evaluation of potential differential cocaine-related effects on receptive and expressive language. A latent construct approach was selected instead of utilizing the expressive and receptive standard scores to allow for consideration of the shared variability within the subtests, strengthening the cohesive measurement of each construct. The independent influence of prenatal cocaine exposure on language was considered after adjusting for child sex and test age and after considering fetal growth (measured by a latent construct of birth weight, head circumference, and length) and gestational age as intercorrelated response variables. The model was also extended to include the McCarthy general cognitive index (GCI) as a correlated response variable to test whether any observed cocaine-associated deficit in language processing was independent of global cognitive functioning. A final series of models included other important sociodemographic covariates.

**Results**

**Sample Characteristics**

A total of 424 children (226 cocaine exposed and 198 non–cocaine exposed) had complete language test results and were included in the SEM analyses. Attrition analyses showed no differences between the 424 cases included and the 52 cases excluded with respect to

2A priori cutoffs represent approximate thirds of the distribution from the originating birth sample of 476, with the condition of a minimum of 30 infants per cell.
study group status, maternal characteristics at delivery (age, prenatal care, primigravida, marital status, employment, and education), and infant characteristics (birth growth parameters, gestational age, and sex). Table I presents a description of selected maternal and child characteristics and maternal drug use patterns during pregnancy measured at birth, including the key covariates introduced into the analyses. Table II depicts selected social and demographic characteristics of the primary caregiver and child measured at the 3-year preschool visit. Table III summarizes mean CELF-P subscale and composite standard scores by group. As described in the Statistical Analyses section, the receptive and expressive subscale scores were used to construct latent variables of expressive and receptive language within the context of the SEM analyses.

**Structural Equation Modeling of Expressive and Receptive Language at 3 Years**

An initial baseline model was constructed, regressing the latent variable constructs of expressive and receptive language as intercorrelated response variables on level of prenatal cocaine exposure, measured by a summary latent construct including information from maternal self-report and toxicology assays. This model also included a latent construct for fetal growth and gestational age as separate intercorrelated response variables, due to the documented influence of prenatal cocaine exposure on intrauterine growth in these full-term infants participating in the Miami PCS (Bandstra et al., 2001) as well as other cohorts. The model was also adjusted for child sex and test age. Results indicated a cocaine-associated decrement for expressive language (Table IV, Model 1). For every SD unit increase in the level of prenatal cocaine exposure, there was a corresponding .168 SD decrease in expressive language functioning (95% CI = –.320, –.015; \( p = .031 \)). The relationship between receptive language and prenatal cocaine exposure was less pronounced and did not meet the bounds of conventional significance.

Model 2 extended Model 1 to include cognitive functioning measured by the McCarthy GCI as an intercorrelated outcome, evaluating the influence of prenatal cocaine exposure on language functioning independent of cognitive ability (which was not related to prenatal cocaine exposure in this model). Results were consistent with Model 1, suggesting a cocaine-specific decrement in expressive language (Table IV, Model 2: expressive language = –.173, 95% CI = –.330, –.016; \( p = .031 \)). A separate model substituting the McCarthy verbal scale for the McCarthy GCI yielded essentially the same results (not shown), substantiating the CELF-P as a strong independent measure of language functioning. The expressive language decrement associated with increased magnitude of cocaine exposure remained stable in a subsequent model with additional statistical

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**Table II. Child Characteristics Assessed at the 3-Year Assessment Visit**

<table>
<thead>
<tr>
<th>Covariate</th>
<th>Non-Cocaine-Exposed (n = 198)</th>
<th>Cocaine-Exposed (n = 226)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Child’s exam age, mos, M (SD)</td>
<td>198</td>
<td>226</td>
</tr>
<tr>
<td>Biological mother as caregiver*</td>
<td>198</td>
<td>224</td>
</tr>
<tr>
<td>Child’s McCarthy GCI score, M (SD)</td>
<td>198</td>
<td>226</td>
</tr>
<tr>
<td>Child bilateral hearing deficit</td>
<td>194</td>
<td>215</td>
</tr>
<tr>
<td>Child blood lead ≥ 10 µg/dl*</td>
<td>189</td>
<td>215</td>
</tr>
<tr>
<td>Caregiver’s education, y, M (SD)</td>
<td>198</td>
<td>224</td>
</tr>
<tr>
<td>Father presence in household*</td>
<td>191</td>
<td>223</td>
</tr>
</tbody>
</table>

GCI = general cognitive index.
*Group n for covariates include only those attending the preschool visit with available language and covariate data.
*p < .05, **p < .01 for between-group comparisons.

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**Table III. CELF-P Standardized Language Scores at 3-Year Preschool Visit**

<table>
<thead>
<tr>
<th>CELF-P Subscale and Composite Standard Scores</th>
<th>Non-Cocaine-Exposed (n = 198)</th>
<th>Cocaine-Exposed (n = 226)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Expressive composite</td>
<td>78.8 (12.7)</td>
<td>76.3 (13.2)</td>
</tr>
<tr>
<td>Recalling sentences in context</td>
<td>7.1 (3.0)</td>
<td>6.4 (3.2)</td>
</tr>
<tr>
<td>Formulating labels</td>
<td>6.0 (2.0)</td>
<td>5.7 (1.9)</td>
</tr>
<tr>
<td>Word structure</td>
<td>5.9 (2.6)</td>
<td>5.4 (2.5)</td>
</tr>
<tr>
<td>Receptive composite</td>
<td>73.1 (11.9)</td>
<td>71.3 (11.0)</td>
</tr>
<tr>
<td>Linguistic concepts</td>
<td>4.9 (2.1)</td>
<td>4.9 (2.2)</td>
</tr>
<tr>
<td>Basic concepts</td>
<td>4.9 (2.2)</td>
<td>4.4 (2.0)</td>
</tr>
<tr>
<td>Sentence structure</td>
<td>5.9 (2.3)</td>
<td>5.5 (2.0)</td>
</tr>
<tr>
<td>Total language composite</td>
<td>75.5 (9.9)</td>
<td>73.0 (9.6)</td>
</tr>
</tbody>
</table>

CELF-P = Clinical Evaluation of Language Fundamentals–Preschool.
adjustment for maternal age and prenatal exposures to alcohol, tobacco, and marijuana (measured by self-report composites for total pregnancy usage as described in the Methods section) (Table IV, Model 3: expressive language $\beta = -.173$, 95% CI $= -.330, -.016$; $p = .031$). An additional model, conducted to check for potential confounding related to blood lead levels ($< 10$ vs. $\geq 10$ mg/dl), hearing abnormalities (expressed as normal in at least one ear vs. bilaterally abnormal), and biological mother caregiving (yes/no), yielded little change in the cocaine-associated decrement in expressive language functioning (Table IV, Model 4: expressive language $\beta = -.213$, 95% CI $= -.423, -.003$; $p = .047$). A final model including caregiver education and father involvement yielded similar estimates to those of the previous models, although the $p$-value was slightly beyond conventional significance levels (Model 5: expressive language $\beta = -.178$, 95% CI $= -.364, .008$; $p = .060$). Estimates for receptive language functioning are also presented in Table IV.

**Discussion**

Cocaine-exposed children at age 3 years exhibited an incremental decrement in expressive language functioning with increasing severity of prenatal cocaine exposure. The relationship between severity of prenatal cocaine exposure and receptive language functioning was less pronounced and not statistically significant in any of the models tested. Expressive and receptive language abilities were evaluated within a statistical model that considered the shared variability between the two constructs and with consideration for other potential correlates of prenatal cocaine exposure and/or language functioning, including fetal growth and child cognitive functioning at age 3. In this context, severity of prenatal cocaine exposure, measured by combining information from quantified maternal self-report and qualitative and quantitative toxicology assays, was consistently related to a decrement in expressive language functioning independent of cocaine’s influence on
fetal growth, gestational age, and global cognitive functioning at age 3 years.

These results remained highly stable after controlling for (1) maternal age and prenatal exposure to alcohol, marijuana, and tobacco, (2) biological mother as the primary caregiver at age 3, and (3) hearing abnormalities and environmental lead exposure. The sample was originally drawn from urban inner-city zip codes, and indicators of socioeconomic status and caregiver education were similar between the groups. In a final model of expressive language functioning that included caregiver education and the father’s presence as a caregiver, there was little evidence of confounding by these two endogenous covariates (i.e., essentially no change in the cocaine effect estimate). The observation of a \( p \)-value slightly beyond conventional significance levels (\( p = .060 \)) may be due to the loss of statistical power and precision when a large number of intercorrelated covariates are included within a model or to qualitative differences in the home environment which may also influence children’s language development. While the current study results support a cocaine-specific influence on expressive language development, it is important to acknowledge that other important developmental and environmental processes may mediate or moderate the influence of prenatal cocaine exposure. For example, language stimulation in the home environment, a potentially important factor in the development of expressive language skills, was not measured at age 3 in this study. However, as noted in another published report from this cohort, the observed relationship between prenatal cocaine exposure and lower total language functioning among cocaine-exposed children through age 7 was independent of home environment measured at age 4\(\frac{1}{2}\) years (Bandstra et al., 2004). Other factors that might be pertinent to early language development, such as receipt of early intervention services, occurred at very low and undifferentiated levels between the groups in the Miami PCS cohort.

The current report augments earlier reports from the same study documenting differences in global language functioning between cocaine-exposed and non-cocaine-exposed children longitudinally from birth through age 7 years (Bandstra et al., 2002; Morrow et al., 2003) and indicates that expressive language functioning may be differentially influenced as a function of severity of prenatal cocaine exposure. Cocaine-exposed children may have greater difficulty with the rules of language governing the production aspects of language processing, over and above that which may be shared in the standardized measurement of receptive and expressive language abilities. These findings, in conjunction with several other studies suggesting impaired expressive language functioning in cocaine-exposed children (Koren et al., 1998; Nulman et al., 2001; Singer, Hawkins, Huang, Davillier, & Baley, 2001), lay the groundwork for more focused investigation of specific language processing modalities. While the present study did not confirm a parallel decrement in receptive language processing abilities measured by the CELF-P in relation to the severity of cocaine exposure, several other studies have observed impairments in receptive language skills associated with prenatal cocaine exposure (Bender et al., 1995; Koren et al., 1998; Nulman et al., 2001), indicating a continued need to disentangle disparities in study results with a greater focus on specific aspects of language processing in prospective, well-controlled studies with adequate sample sizes to detect results.

The measurement of expressive and receptive language, or speaking and listening skills, respectively, is a common practice in standardized testing but tends to distill the numerous processing aspects of language into broad test-specific definitions that often overlap and interact at the level of test performance (Moats, 1994). Moats (1994) argues that a more meaningful distinction should focus on specific language systems, including phonology, morphology, syntax, and semantic organization. While standardized assessments such as the one utilized in the present study are highly beneficial in ensuring common data collection procedures for research purposes, standardized language tests do not sample all aspects of language and may not be sensitive to subtle variations in discourse, pragmatics, syntax, and semantics. Preliminary work in this area with small samples has documented that cocaine-exposed children are more likely to exhibit phonological delay (Madison, Johnson, Seikel, Arnold, & Schultheis, 1998), poorer quality of discourse pragmatics and delayed syntactic development (Mentis & Lundgren, 1995), less complex language skills (Malakoff et al., 1999), and delayed and restricted semantic representations (Bland-Stewart, Seymour, Beeghly, & Frank, 1998) when compared with non-cocaine-exposed children. This level of detailed information is important in guiding future research directions and designing effective intervention strategies that might benefit children with prenatal cocaine exposure.

In the current study, effect estimates summarizing the relationship between prenatal cocaine exposure and expressive language abilities suggested an approximate one-fifth SD decrease in expressive language functioning
for each unit increase in the magnitude of prenatal cocaine exposure. This represents a subtle gradient of effect, but one with potential economic ramifications for service providers, as the associated distributional shift in language functioning may result in an increased number of cocaine-exposed children qualifying for language-based special education services (Lester et al., 1998). For example, 39% of the cocaine-exposed children in the current study fell below the standard language cutoff score of 70 used to determine service eligibility, as opposed to 28% of non-cocaine-exposed children. While both groups had higher than expected percentages of children potentially in need of language remediation, cocaine-exposed children were clearly at increased clinical risk. This is consistent with a recent report by Delaney-Black and colleagues (2000) indicating that cocaine-exposed children were two and a half times more likely to be categorized as low language functioning than were non-cocaine-exposed control children at age 6 years, yet no mean differences were observed between the two groups on language abilities measured by a standardized language scale or with respect to coded language samples.

The current study utilized a multivariate strategy to characterize severity of exposure, combining all available self-report and bioassay information. Quantifying degree of prenatal substance exposure is an extremely complex issue. With only recent attention in the published literature and no agreed-upon methodology for establishing a common measurement approach (Carmichael Olson & Toth, 1999), comparisons across studies remain limited. In addition, few studies have specifically evaluated severity of exposure in relation to language functioning. One study, using measures culled from maternal self-report, reported no associated decrement in language functioning in relation to severity of cocaine exposure (Hurt et al., 1997). Conversely, Singer, Arendt, and colleagues (2001) found that more heavily exposed infants (defined by maternal self-report > 70th percentile or > 75th percentile of meconium benzoylecgonine concentration) had lower auditory comprehension scores than the nonexposed infants, and lower total language scores than the more lightly exposed and nonexposed infants. In a separate report from the Miami PCS, longitudinal growth curve modeling of the latent construct for cocaine severity was related to global language functioning at 3, 5, and 7 years (Bandstra et al., 2004). While the approach used in the Miami PCS will need to be replicated and validated in future work, it substantiates several recent studies indicating modest relationships between heavier degrees of prenatal cocaine exposure and various other infant and child developmental outcomes (Bateman & Chiriboga, 2000; Delaney-Black et al., 1996; Eyer, Behnke, Conlon, Woods, & Wobie, 1998; Frank et al., 1999; Kuhn, Kline, Levin, & Susser, 2000; Tronick, Frank, Cabral, Mirochnick, & Zuckerman, 1996). From a teratological perspective, it is important to identify whether the influences of prenatal cocaine exposure can be differentiated based upon a gradient of exposure. Another possibility is a nonlinear-threshold relationship without a gradient. Whereas this study’s methods included combined self-report and bioassay information, additional work is needed to validate the best approaches to defining severity of exposure more consistently across studies (Carmichael Olson & Toth, 1999).

Several additional characteristics of the current study should be noted when interpreting its results. The study cohort was intentionally restricted at enrollment to full-term, healthy African American infants born to mothers residing in socially disadvantaged inner-city neighborhoods. This sampling approach controlled for many of the demographic, health, and social conditions that might influence child development, increasing the study’s capacity to draw conclusions about the influence of prenatal cocaine exposure when evaluating multidetermined, complex child outcomes. Importantly, African American children living in low-income homes are known to be at increased risk for showing difficulties with many of the early language skills associated with literacy. While much work remains to elucidate the mechanisms for this trend, the effects of poverty perse, impoverished early home literacy experiences, and variations in African American English dialectical speech are believed to be important contributing factors (Washington, 2001). Results from the current study suggest that African American children exposed prenatally to cocaine are at increased risk for expressive language deficits when compared with a racially and demographically similar non-cocaine-exposed control group; however, these findings may be generalized only to similar populations and urban settings.

It is plausible that the mechanisms by which cocaine influences child development may differ in samples of children with more varied risk levels, different racial/ethnic backgrounds, premature birth, or other health conditions. This is evident in a study by Singer, Hawkins, and colleagues (2001), in which a dramatic 13-point mean difference in expressive language functioning was observed between cocaine-exposed and non-cocaine-exposed very-low-birth-weight children. It
should also be noted that preschool assessment instruments are known to be some of the least stable child measures, in part due to the difficulty of assessing very young children, who may be more inhibited in the formal testing environment. Findings such as those reported in the current study will need to be confirmed among older children to evaluate the long-term stability and impact of subtle differences in language processing abilities. Finally, the veracity of self-report methodologies for collecting substance use information is difficult to ascertain. Although the present study was strengthened by the use of a combination of self-report and bio-assay methodologies to determine prenatal exposure to cocaine and other drugs, classification errors may still have occurred, with potential biases that might have drawn the study estimates to the null.

The present report indicated a subtle gradient relationship between the magnitude of prenatal cocaine exposure and expressive language abilities in a large, well-retained cohort of children enrolled prospectively at birth. Further study of the home and child-rearing environment is needed to better understand both the prenatal and postnatal etiological context in which the expression of cocaine-related effects occurs across the developmental continuum. Finally, the importance of early language development to later academic success, particularly in the areas of reading and writing, is well established. The findings from this and other studies suggest that cocaine-exposed children may be at increased risk for subtle language-based deficits that may have important ramifications for their long-term academic and social adaptation, particularly when taken into consideration within the broader context of other risk factors and causal determinants of academic success.

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