Language Production in 24-Month-Old Inner-City Children of Cocaine-and-Other-Drug-Using Mothers

MARGUERITE E. MALAKOFF
Harvey Mudd College

LINDA C. MAYES
Yale University School of Medicine, Yale Child Study Center

RICHARD SCHOTTENFELD
Yale University School of Medicine, Department of Psychiatry

STEVEN HOWELL
Yale University School of Medicine, Yale Child Study Center

Language abilities of 46 inner-city children exposed to cocaine in utero and 28 non-cocaine-exposed inner-city peers, all in the continuing care of their biological mothers, were compared at 24 months of age. The sample was recruited prior to or at the birth of the target child, and had been followed for a minimum of 24 months. Data on drug use during pregnancy were available for all mothers; data were also collected on postpartum drug use. Natural language data were collected during a play session between child and caregiver. Results revealed significant effects for *exposure* and *gender* on measures of language complexity. Non-cocaine-exposed toddlers produced more complex language than did their exposed peers, and girls produced more complex language than did boys. Possible relations to environmental conditions and implications for future outcomes are discussed.

Within the last decade, many investigators have focused on the potential physical, developmental, and psychological effects of prenatal cocaine and other drug exposure on infants and young children. These studies have utilized a variety of designs, including longitudinal, cross-sectional, case-control, and retrospective cohorts; they

Direct all correspondence to: Dr. Marguerite Malakoff, Department of Humanities and Social Sciences, Harvey Mudd College, 301 E. 12th St., Claremont, CA 91711-5990. E-mail: <margo_malakoff@hmc.edu>.

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have defined in multiple ways the independent variable(s) (i.e., the amount and duration of cocaine as well as other drug exposure), and they have emphasized a host of different outcomes (Neuspiel, 1995; Lester, Freier, & LaGasse, 1995; Brooks-Gunn, McCarton, & Hawley, 1994). Although still scant, inconsistent, or inconclusive on many crucial issues and marked by a number of methodological problems, published studies to date nonetheless reveal the beginnings of a profile of possible cocaine-related effects on neuropsychological functions involved in the regulation of arousal and attention and reactivity to stressful conditions (Azuma & Chasnoff, 1993; Mayes & Bornstein, 1995; Frank, Bresnahan, & Zuckerman, 1993; Richardson, Day, & McGauhey, 1993; Singer, Farkas, & Kliegman, 1992). Findings reported across several studies include increased behavioral lability, impulsivity, diminished novel exploration, and diminished cortisol response (e.g., Alessandri, Sullivan, Imaizumi, & Lewis, 1993; Griffith, Azuma, & Chasnoff, 1994; Mangano, Gardner, & Karmel, 1992; Mayes, Bornstein, Chawarska, Hynes, & Granger, 1996).

Concerns have also been raised regarding the impact of maternal drug use on the adequacy of the child's caregiving environment (Hawley, Halle, Drasin, & Thomas, 1995; Kronstadt, 1991; Malakoff, Mayes, & Schottenfeld, 1994; Rotholz, Snyder, & Peters, 1995; van Barr & de Graaff, 1994). Indeed, several investigators have suggested that the impact of perinatal substance use on the child's caregiving environment may convey the greater risk for delayed and impaired social-emotional, language, and cognitive development in children, above and beyond their prenatal exposure status (Alessandri, Sullivan, Bendersky, & Lewis, 1995; Lester et al., 1995; Mayes, 1995; Pawl, 1992; Woods, Behnke, Eyler, Conlon, & Wobier, 1995).

Early language development could be affected by maternal cocaine use through at least three different and potentially interactive pathways. First, the effect of prenatal cocaine exposure on the regulation of attention and arousal may have important developmental implications for language acquisition. Successful language acquisition requires that infants attend to and process the specific language input in their environment. Even a subtle disregulation of attentional systems could potentially disrupt an infant's ability to extract and process the linguistic information available in the environment.

Second, cocaine and other drug use can disrupt parental interactions with their infant. Research in developmental psycholinguistics has established the importance of caregiver–child linguistic interactions in early language acquisition. Poor linguistic stimulation and social isolation in the infant and toddler years have been associated with language delays (Bee et al., 1982). Features of maternal linguistic style that have been found to be positively correlated with child language development include a high total volume of child-directed speech and a high rate of contingent responding (Hoff-Ginsberg & Shatz, 1982; Pine, 1994). Language development has especially been associated with mothers' responsiveness to their children's utterances (Hoffer & Bliss, 1990; Huttenlocher et al., 1991; Morisset, Barnard, & Booth, 1995), with the amount of time mothers spend interacting with their children, and with the conversational nature of child-directed speech (Hoff-Ginsberg, 1991; Tomassello & Farrar, 1986).

The quality of dyadic interactions is of particular concern among drug-using parents and their children (Mayes, 1995). Some findings suggest that cocaine-using

parents interact less with their infants and rely less on verbalization as a means of engaging the infants' attention (Burns, Chetnik, Burns, & Clark, 1991; Mayes, Feldman, Granger, Haynes, Bornstein, & Schottenfeld, 1997; Gottwald & Thurman, 1994). The effects of cocaine use on responsiveness may decrease not only the quality of the linguistic interaction, but also the variety and richness of the linguistic interactions to which infants and toddlers are exposed.

A third pathway is the larger social environment in which caregiving occurs. The effects of the social environment on language development are evidenced by the consistent findings that children of lower socioeconomic status (SES) have less advanced language skills than do higher SES children. These differences have been found in measures of vocabulary size, sentence complexity, communication skills, metalinguistic ability, and reading achievement (Deutsch, 1965; Greenwood et al., 1992; Hart & Risley, 1995; Hoff-Ginsberg, 1991; Shore, 1995; Ravid, 1995; Snow, 1983; van der Geest, Gerstel, Appel, & Tervoort, 1973; Walker, Greenwood, Hart, & Carta, 1994; Whitehurst, 1997). Cocaine use among inner-city women is associated with an increased incidence of early school drop-out and poor school performance, parental psychopathology including maternal depression (Latkin & Mandell, 1993; Neuspiel & Hamel, 1992; Mirin, Weiss, Griffin, & Michael, 1991; Rounsaville et al., 1991; Strickland, James, Myers, & Lawson, 1993), homelessness and family disruption, and domestic violence (Boyd, 1993; Boyd & Mieczkowski, 1990; Lawson & Wilson, 1980; Mayes, 1995).

Only a few published studies have focused on relations between prenatal cocaine and other drug exposure on language development, and the findings have been mixed. A longitudinal study of 35 drug-exposed children in Amsterdam (van Barr, 1990; van Barr & de Graaff, 1994) found delays in early language development, which were still evident at 5 years of age. However, most of the drug-using mothers in the study were polydrug users and their children had been exposed prenatally to a combination of methadone, heroin and cocaine, making it difficult to delineate the effects, if any, of specific drugs. Angelilli et al. (1994), in a retrospective study of 29 2–4-year-old preschoolers, found that language-delayed preschoolers were more likely to have a history of drug exposure than were non-language-delayed same-age peers. However, because of its retrospective nature, this study could not address the likelihood of cocaine-exposed infants developing language delays. The study also did not control for potential confounding variables in the prenatal and postnatal environments, making it difficult to establish the nature of the relationship between prenatal cocaine-exposure and early language delays.

Griffith, Azuma, and Chasnoff (1994) reported that at age 3, children in both polydrug/cocaine exposed and polydrug/no-cocaine-exposed groups showed significantly lower scores than did a drug-free control group on the verbal reasoning subscale of the Stanford-Binet Intelligence Scale. The authors also found that within the polydrug/cocaine-exposed group, the children who continued to live in a drug-using household after birth were more likely to show a delay of one or more standard deviations on the subscale of verbal reasoning than were the children living with a drug-free relative.

Rotholz and co-workers (1995) and Hawley and colleagues (1995) both found no effect of prenatal cocaine exposure on early language development among 2–5-

year-olds. However, the wide age range of the sample in both studies precludes comparisons of early and later language development. Malakoff and colleagues (1994) also found no effects for prenatal exposure on the language abilities of 2½–5½-year-old preschoolers living with cocaine- and other drug-using mothers. Although the majority of the children showed serious language delays, language abilities of youngsters who were exposed prenatally to cocaine did not differ from those whose mothers began using cocaine while the children were infants. This study also revealed a great heterogeneity in outcome, as has often been noted among cocaine-exposed children (van Baar & de Graaff, 1994; Malakoff et al., 1994).

The purpose of the present study was to examine the development of language production among 24-month old infants exposed to cocaine in utero and in the continuing care of their biological mothers, after controlling statistically for potentially relevant perinatal and demographic variables and for polydrug use. Specifically, the focus was on grammatical complexity, lexical diversity, and grammatical diversity as measures of early language development. A comparison group of inner-city non-drug-using mothers and their infants were included to control for socioeconomic variables, and extensive background information, including drug, alcohol, and cigarette use, was collected from all participants. To obtain naturalistic language samples from both members of the dyad, language was recorded during a play session between the child and parent.

Methods

Sample

A sample of 74 infants (46 cocaine- and other drug-exposed and 28 non-cocaine-exposed) were randomly selected from a large longitudinal study of the effects of prenatal cocaine exposure on infant and child development. The mothers were invited to join the larger study when they presented for prenatal care at the Women's Center of Yale–New Haven Hospital or, in the case of no prenatal care, when they were admitted to the postpartum ward. After obtaining verbal consent for an interview, all women were questioned about substance use using a detailed interview that covered lifetime use of cocaine, tobacco, alcohol, marijuana, and other drugs (e.g., sedative, opiates), and the frequency and amount of use of these agents during the preceding 30 days. The interview was administered by trained research interviewers. In the state of Connecticut in the time covered by this study, maternal drug use did not automatically require a referral to child protective services. Every women arriving for prenatal care was interviewed about HIV status, and HIV antibody testing was offered to all women, regardless of drug use.

For all women, regardless of drug-use history, a urine sample was obtained for toxicology and was screened for cocaine, heroin, benzodiazepine, and tetrahydrocannabinol (cannabis). Maternal cocaine exposure status was determined either by self-report of use during pregnancy or by a positive urine screen at a prenatal visit or at delivery. Nonexposed status was ascertained by maternal and infant urine toxicology and a negative maternal history of cocaine use during pregnancy and at the time of delivery.

For purposes of this study: although polydrug use was involved, these mothers hereafter are referred to as "cocaine-using" and their children as "cocaine-exposed."

For those women who used cocaine, the mean number of years of cocaine use was 4.33 (SD = 3.6), with a range of 0–14 years. Significant differences existed between cocaine-using and non-cocaine-using women in the use of alcohol and tobacco. Fifty-nine percent (n = 27) of the cocaine-using mothers and 17% (n = 5) of the non-cocaine-using mothers reported at least 1 or more days of alcohol use in the last month during their pregnancy, $\chi^2 = 11.83$, $p < .001$. For those mothers reporting alcohol use in the previous 30 days, the average number of days of use was 5.8 (SD = 7.1) for cocaine-using and 1 for the two non-cocaine-using women. Cocaine-using women who used alcohol reported on average 2.64 (SD = 1.95) drinks per use compared with 1 for the two non-cocaine-using women. Cigarette smoking was reported by 98% of cocaine-using mothers (n = 41), whereas 29% (n = 8) of non-cocaine-using mothers reported at least 1 or more days of tobacco use in the previous 30 days ($\chi^2 = 28.53$, $p < .0001$). The average amount smoked was 1.85 (SD = 1.4) packs per day in the previous 30 days for the cocaine-using and 2.0 (SD = 1.3) for the non-cocaine-using group. Twenty-two percent of the cocaine-using (n = 10) and 11% (n = 3) of the non-cocaine-using mothers reported at least 1 day of marijuana use in the last month during their pregnancy ($\chi^2 = 1.46$, ns).

Mothers in both groups were asked to participate in a study about early cognitive and social development of infants and young children. Mothers of the cocaine-using group were aware that the investigators knew about their drug history and consented to have their infants followed at 6-month intervals independent of their own participation in any drug-treatment intervention services. All infants in this sample remained in their mothers' care after delivery. Although detailed information on caretaking history was not available, it should be noted that children of cocaine-using parents are more likely to have multiple caretakers within the family owing to the limited parental caretaking abilities related in part to the severity of their addiction.

Follow-up data on drug-use was obtained through the treatment program in which drug-using mothers were participating. Interviews regarding substance use were conducted at 6-month intervals following delivery and were accomplished using the Addiction Severity Index (ASI); (McLellan et al., 1992). The ASI data for this cohort of drug-using women revealed that 84.8% reported continued drug use (alcohol, tobacco, marijuana, and/or cocaine) at all follow-up visits for 2 years after the birth of the target child; only 2.2% of the women had discontinued drug use. Data were missing for the remaining 13% of the drug-using group.

Continued use of cocaine showed similar results: 78.3% of the women continued to use crack-cocaine until the 24-month follow-up visit, whereas only 8.7% (n = 4) women had discontinued or decreased their use since the birth of their child. Furthermore, the results revealed that no woman who reported drug use after the birth of the child stopped drug use completely by the 24-month follow-up visit.

Table 1 shows the demographic characteristics of mothers and infants in the cocaine- and non-cocaine-exposed groups at the end of pregnancy. The sample was predominantly African American (85% cocaine-exposed and 82% non-cocaine-exposed). Most women were in their 20s; however, cocaine-using mothers were

Table 1. Sample Characteristics by Group: Cocaine-Exposed and Non-Cocaine-Exposed Group

Characteristics	*Non-Cocaine-Exposed* (n = *28*)	*Cocaine-Exposed* (n = *46*)	$F/\chi^2(G^2)$
Maternal			
Age (years), mean (SD)	24.9 (3.6)	28.5 (4.6)	12.74***
Completed high school	71%	60%	0.32
Single mother	67%	76%	2.02
Alcohol used in last month of pregnancy	17%	59%	11.85***
Marijuana used in last month of pregnancy	11%	22%	1.46
Tobacco used during pregnancy	28%	98%	28.53***
Race			7.87 (G^2)
African American	82%	85%	
Caucasian	4%	13%	
Hispanic	4%	2%	
Asian	4%		
Child			
Birth weight (g)	3302.71	2692.44	21.28***
Birth length (cm)	51.21	47.07	10.72**
Head circumference (cm) (controlled for birth weight)	34.03	32.59	7.56**
Mean gestational age (wks), mean (SD)	39.5 (1.3)	38.2 (2.5)	6.59*
Premature (= 36 wks)	7.1%	23.9%	3.388
Small for gestational age	3.7%	12.2%	3.39 (G^2)
OCS, mean (SD)	100.82 (21.21)	79.16	18.46***
Bayley Scales, mean (SD)			
MDI	84.60 (13.46)	78.36 (12.06)	3.918 (*df* = 1,67)
PDI	98.56 (11.96)	91.39 (15.0)	4.19*** (*df* = 1,67)

(*df*) Included to indicate missing data; G^2 = Likelihood Ratio; $p > .10$ unless noted otherwise with asterisks.
* $p < .05$. ** $p < .01$. *** $p < .001$.

significantly older than were non-cocaine-exposed mothers, $F(1, 71) = 12.74$, $p < .001$. The majority of the women in both groups were single mothers. A greater proportion of women in the non-cocaine-using group (71%) had completed high school than women in the cocaine-using group (60%), although this difference did not achieve significance. More detailed educational information was available for 71% of the non-cocaine-using mothers and 76% of the cocaine-using mothers; there was no significant relationship between group and level of education achieved. All of these women had received at least some high school education, and 35% of the non-cocaine-using mothers and 20% of the cocaine-using mothers had some college experience; however, only 5% of the non-cocaine-using mothers and 5.7% of the cocaine-using mothers had completed a college degree. There were no differences in the proportion of mothers in each group receiving prenatal care, and the majority of women in both groups had at least one prenatal visit.

Mean gestational age was younger for infants of cocaine-using mothers than for those of non-cocaine-using mothers, $F(1, 71) = 6.59$, $p < .05$. Cocaine-exposed groups showed lower birthweight, $F(1, 71) = 21.28$, $p < .0001$, and length, $F(1, 69) = 10.72$, $p < .005$, but no difference in head circumference when controlling for birthweight. The prenatal and delivery course was summarized at delivery using the Obstetric Complications Scale (OCS) (Littman & Parmelee, 1978), a measure of the number of optimal conditions during a pregnancy and delivery. Cocaine-exposed infants showed significantly worse OCS scores, with a higher score indicating more complications during gestation, $F(1, 70) = 18.46$, $p < .0001$. There were six infants born small for gestational age (SGA), which is defined as birthweight greater than two standard deviations below that expected for gestational age. Five of these infants were in the cocaine-exposed group, accounting for 12.2% of the cocaine-exposed infants compared with 3.7% of the non-cocaine-exposed infants, $G^2 = 3.39^1$, *ns*. Thirteen infants were born between 33–36 weeks gestational age, 11 of whom were in the cocaine-exposed group, accounting for 23.9% of the cocaine-exposed infants compared with 7.1% of the non-cocaine exposed infants, $G^2 = 3.38$, $p < .07$. When applicable, the age at which children were seen was corrected for gestational age when less than 36 weeks.

There was no difference between the groups in mean age (calculated in days) when the two groups were seen at 24 months. The mean age of the cocaine-exposed group was 24 months, 5 days ($SD = 20.1$ days), with a range of 22.9–26.1 months. The mean age of the non-cocaine-exposed group was 24 months, 4 days ($SD = 23.2$ days), with a range of 22.9–26.8 months. There were 19 boys (41%) and 27 girls (59%) in the cocaine-exposed group and 10 boys (36%) and 18 girls (64%) in the non-cocaine-exposed group. All children were accompanied by mothers.

General developmental status was assessed at 24 months using the Bayley Scales of Infant Development (1993). The difference between the two groups on the composite index of mental development (MDI) was marginally significant, with the non-cocaine-exposed toddlers showing a higher mean score ($F(1, 70) = 3.91$; $p < .06$). Cocaine-exposed children showed a significantly lower mean score on the motor scale (PDI), $F(1, 67) = 4.19$, $p < .05$.

Procedures

Natural language data were transcribed from a videotaped semi-structured play at the 24-month follow-up visit. The sessions ranged 4–8 minutes in length. The majority of the sessions (74%) were between 4.5–6.5 minutes long; only 8% were under 4.5 minutes. The mean length of the play session was similar for both groups, with a mean of 5.6 minutes (SD = .94).

All play sessions took place in the same room and with the same set of toys. The caregiver and child were accompanied to a small room with a one-way mirror permitting observation and videotaping. Toys, which included a teddy bear-like doll, a tea-set, blocks, a blanket, a train, a doll, set of stacking barrels, two picture-only books, a small ball, and a toy phone, were laid out visibly on the floor at one end of the room. Other furnishings in the room included a small chair and table and a quilted mat for the child to play on. Sessions were minimally 4 minutes in length and were at the end of the visiting period. After a brief background survey administered by an investigator, the caregiver was asked to play with the child as she or he normally would, and the two were left alone in the room until an investigator terminated the session. Some sessions extended longer than the minimum 4 minutes when mother and child continued to play past the ending time of the visit.

The data were transcribed, coded, and analyzed using the CHILDES language analysis system (MacWhinney, 1995). Trained research assistants who were blind to group assignment transcribed the session using in alternation both audiotapes and videotapes. Two independent research assistants compared the transcription to the videotape and resolved a small number of discrepancies through consensus. Language measures were calculated separately for caregiver and for child.

Measures

Measures of caregiver and child speech were assessed to reflect rate of speech, grammatical complexity, richness of vocabulary, and richness of grammatical categories. Rate of speech was calculated as the total number of utterances produced during the session, divided by the length of the session. Grammatical complexity was assessed with three measures, all based on complete and intelligible utterances produced. *Mean length of utterance* (MLU) in morphemes has become the standard for measuring children's average utterance length. *Mean of the five longest utterances* (MLU5), calculated as the average length of the five longest utterances measured in morphemes, reflects the upper limits of the grammatical complexity produced. The third measure, *Mean words/utterance* (MWU), the mean length of utterance measured in words, was computed to control for possible dialectical differences. Of special concern were differences in morphemization between Ebonics and Standard English that might deflate MLU.

The richness of vocabulary was assessed with two measures. *Type-token ratio* (TTR), a widely used measure of lexical diversity, is computed by dividing the number of different word roots, ignoring pluralization and tense, by the total number of words (*cat* and *cats* are a single word type, as are *look*, *looks*, and *looked*).

Because it is a ratio score, TTR is strongly affected by the total number of words uttered and reflects both amount and variety of speech. *Number of different word roots* is a frequency count of the total number of different word roots produced, ignoring pluralization and tense, divided by the session length in minutes to control for time. This measure is less sensitive to the total number of words uttered than is TTR, and it reflects the breadth of vocabulary over a given span or time. Richness of grammatical productions was calculated as the *percentage of speech* falling within five categories: verbs, nouns, modifiers (adjectives and nouns), functors (e.g., prepositions, pronouns, articles, negators), and communicators (a variety of words and pseudo-words that serve to support the interaction; e.g., please, thank you, play sounds, interjections, family words, and nonwords).

Measure Intercorrelations

All measures, with exception of TTR, were moderately to strongly intercorrelated. As anticipated, MLU and MWU were highly correlated ($r = .97$). The MLU5 was also strongly correlated with MLU ($r = .75, p < .001$) and MWU ($r = .80, p < .001$). The TTR showed only a moderate correlation with *number of different word roots* ($r = -.25, p < .05$), underscoring the difference between the two measures. *Number of different word roots* was moderately correlated with MLU ($r = .39, p < .001$), MLU5 ($r = .65, p < .001$), and MWU ($r = .44, p < .001$). The TTR also showed a significant correlation with MLU5 ($r = -.30, p < .01$).

Results

The length of the play session showed no significant main effects or interaction effects for *exposure* and *gender*. The number of words children produced during a play session ranged from 3 to 212, with a mean of 68 words (12 words/minute; $SD = 6.2$). Caregivers produced between 144 and 732 words, with a mean of 367 words (65.7 words/minutes; $SD = 17.3$). There were no *exposure* or *gender* main effects for the amount of speech produced, either when compared as total speech or rate of speech. Neither session age nor session length was significantly correlated with the language variables analyzed.

All child outcome variables were entered in a two-way analysis of covariance (ANCOVA), with covariates entered first. Background variables were included as covariates if associated with any of the outcome measures at $p < .1$. The included covariates were *birthweight* (grams), *head circumference* (centimeters). Although not significantly correlated with the outcome variables, three measures of other-drug use were also covaried for theoretical reasons and based on the fact that they have correlated with outcome in other studies from this sample (Mayes et al., 1997). These drug measures were *alcohol* (whether alcohol was used in the last 30 days of pregnancy), *cigarettes* (whether cigarettes were smoked in the last 30 days of pregnancy), and *marijuana* (whether marijuana was used in the last 30 days of pregnancy). *Exposure* and *gender* were entered as between-subject factors. Table 2 presents the correlation matrix for the covaried and outcome variables.

Table 2. Pearson *r* for Intercorrelations Between Covariates Included in the ANCOVA and Child Outcome Variables

	Head Circumference	*Birth Weight*	*Substance Abuse*		
			Alcohol	*Marijuana*	*Cigarettes*
MLU	0.20°	0.21°	−0.10	0.11	−0.01
Mean words/utterance	0.24*	0.23*	−0.08	0.14	−0.03
MLU5	0.23°	0.21°	0.05	0.30**	−0.08
Different word roots	0.28*	0.26*	−0.09	0.23*	−0.31**
TTR	−0.12	0.03	−0.03	−0.21°	0.11
Composite language score[a]	0.24*	0.29*	−0.07	0.18	−0.12
% Communicators	−0.29*	−0.32**	0.18	−0.14	0.29*
% Functors	0.22°	0.18	−0.14	0.07	−0.09
% Nouns	0.16	0.17	−0.14	0.04	−0.35**
% Verbs	0.13	0.20°	−0.02	0.11	−0.07
% Modifiers	0.19	0.26*	−0.07	0.17	−0.05

° $p < .10$. * $p < .05$. ** $p < .01$.

[a] Composite language score was calculated by summing the *z*-scores for MLU, MLU5, number different word roots, and TTR.

Child Speech

Rate of Speech. There were no significant main effects, interaction effects, or covariate effects for rate of speech. Children in the non-cocaine-exposed dyads produced a mean of 11.7 words per minute ($SD = 6.7$), whereas children in the cocaine-exposed dyads produced a mean of 13.50 utterances per minute ($SD = 5.3$).

Grammatical complexity. The ANCOVA revealed a single significant covariate effect of *marijuana* for MLU5 ($F(1, 61) = 11.23, p < .005$). A significant main effect emerged for *exposure* for MLU ($F(1, 61) = 8.08, p < .01$) and MWU ($F(1, 61) = 6.25, p < .05$), and there was a marginal effect for MLU5 ($F(1, 61) = 3.48, p < .06$). Cocaine-exposed toddlers produced shorter utterances in terms of morphemes ($M = 1.45$; $SD = .30$) and words ($M = 1.40$; $SD = .28$) than did their non-cocaine exposed peers ($M_{MLU} = 1.67$, $SD_{MWU} = .44$; $M_{MWU} = 1.56$; $SD_{MWU} = .33$). The mean of the five longest utterances (in morphemes) was also longer for cocaine-exposed toddlers ($M = 3.11$; $SD = 1.14$) than among their non-exposed peers ($M = 3.64$; $SD = .1.21$). A main effect for *gender* also emerged for MLU ($F(1, 61) = 6.329, p < .05$), MWU ($F(1, 61) = 6.43, p < .05$), and MLU5 ($F(1, 61) = 4.04, p < .05$). Boys produced shorter utterances both in terms of morphemes ($M = 1.42$; $SD = .33$) and words ($M = 1.36$; $SD = .29$) than did girls ($M_{MLU} = 1.61$; $SD_{MLU} = .38$; $M_{MWU} = 1.52$, $SD_{MWU} = .30$). Table 3 reveals that MLU5 showed a similar pattern of results. Table 3 also reveals that the standard MLU and the modified measure, mean words/utterance, revealed similar results, suggesting that MLU is a reliable measure for this population at this age.

Lexical Diversity. Table 3 reveals a heterogeneity of performance among all groups on the *number of different word roots* produced. However, the pattern of means resembles that found for grammatical complexity, with non-cocaine-exposed

Table 3. Mean, *SD*, and *F*-ratio (Controlling for Birth Weight, Head Circumference, and Other Drug Use) for Children's Grammatical Complexity, Lexical Diversity, and Composite Language Score by Gender for Cocaine-Exposed (CE) and Non-Cocaine-Exposed (NCE) Children

	Boys				*Girls*						
	NCE (n = *10*)		*CE* (n = *18*)		*NCE* (n = *18*)		*CE* (n = *28*)		*Exposure*	*Gender*	*Exposure* × *Gender*
Measure	*Mean*	SD	*Mean*	SD	*Mean*	SD	*Mean*	SD	(F)	(F)	(F)
Rate of speech	13.79	6.31	10.89	6.59	13.38	4.92	12.23	6.64	0.335	1.05	1.02
MLU	1.47	0.32	1.39	0.34	1.78	0.46	1.50	0.27	8.08**	6.33*	1.41
Mean words/utterance	1.38	0.24	1.35	0.32	1.66	0.33	1.43	0.25	6.25*	6.43*	1.94
MLU5	3.14	1.92	2.89	1.24	3.92	1.10	3.25	1.06	3.84°	4.04*	0.42
Different word roots	6.43	2.73	4.87	2.41	6.55	2.28	5.58	2.89	0.02	1.26	0.71
TTR	0.54	0.17	0.57	0.17	0.58	0.14	0.55	0.13	0.11	0.01	0.18
Composite language score[a]	−0.20	3.04	−1.06	2.26	1.58	2.40	−0.282	2.11	3.94°	4.84*	0.32

[a] Composite language score was calculated by summing the *z*-scores for MLU, MLU5, number different word roots, and TTR.
° $p < .10$. * $p < .05$. ** $p < .01$.

girls showing the greatest vocabulary range (M = 6.55) and cocaine-exposed boys the smallest (M = 4.87). The ANCOVA revealed two significant covariates, both for *number of different word roots*: *cigarettes* ($F(1, 61) = 4.29, p < .05$) and *marijuana* ($F(1, 61) = 6.63, p < .05$). *Marijuana* also showed marginal significance for *TTR* ($F(1, 61) = 2.96, p < .1$). No main effects or interaction effects for either number of different word roots or TTR emerged. This lack of difference may be because of the sensitivity of this measure to the total number of words produced. A TTR score of 1 indicates only that every word produced was different from every other word, and therefore children who utter a small number of different words would show a TTR score equivalent or higher to a child who produced more varied speech, but also repeated words more often.

Grammatical Diversity. The grammatical composition of the children's speech is presented in Table 4. Five broad grammatical categories were compared. *Modifiers* combines adjectives and adverbs, both of which were produced with low frequency. *Functors* is a broad category that includes "small" grammatical function words, such as prepositions, pronouns, negators, articles, and wh-words. *Communicators* are words and pseudo-words that serve to sustain the linguistic interaction. Four types of communicators were included: standard "politeness" words (please, thank you, hello); interjections (e.g., huh, oh, hey), onomatopoeic play and animal sounds, and nonwords that were clearly intended to further the linguistic interaction (eh, uh, mm). Unintelligible words were *not* included in this latter category.

Analyses of these variables revealed significant and marginally significant covariates. *Marijuana* emerges as a significant covariate for *percent communicators* ($F(1, 61) = 4.30, p < .05$) and was marginally significant for *percent modifiers* ($F(1, 61) = 3.57, p < .07$. *Cigarettes* emerged as a significant covariate for *percent nouns* ($F(1, 61) = 5.23, p < .05$); and *birthweight* emerged a marginally significant covariate for *percent modifiers* ($F(1, 61) = 3.93, p < .06$).

The ANCOVA revealed a significant main effect of *exposure* for *percent functors* ($F(1, 61) = 4.00, p < .05$). The speech of non-cocaine-exposed children showed greater *percent functors* (M = 24%, SD = 11) than for exposed children (M = 18%; SD = 11). This change is consistent with normal language development, where the use of functors increases while reliance on communicators diminishes with increasing language. Among caregivers, *percent functors* comprised 37% of their speech, whereas *percent communicators* comprised only 10%. Table 4 also shows that exposed children used a greater *percent communicators* (M = 46%; SD = 23) than did non-exposed children (M = 33, SD = 18). It is interesting to note that when covariates were not included, this difference achieved significance ($F(1, 70) = 6.27, p < .05$).

Gender also showed significant main effects for *percent communicators* ($F(1, 61) = 9.31, p < .005$), *percent functors* ($F(1, 61) = 10.75, p < .005$), and *percent nouns* ($F(1, 61) = 4.83, p < .05$). Girls used a greater percentage of functors (M_{GIRLS} = 23%, SD = 11; M_{BOYS} = 16%, SD = 11) and nouns (M_{GIRLS} = 24%, SD = 14; M_{BOYS} = 18%, SD = 11) and relied less on communicators (M_{GIRLS} = 35%, SD = 20; M_{BOYS} = 49%, SD = 23) than did boys. Examination of the means in Table 4 further suggests that non-cocaine-exposed girls are producing the more complex

Table 4. Mean, Standard Deviation, and *F*-ratio (Controlling for Birth Weight, Head Circumference, and Other Drug Use) for Percent of Grammatical Categories in Children's Speech by Gender for Cocaine-Exposed (CE) and Non-Cocaine-Exposed (NCE) Children

	Boys				*Girls*						
Percent of	*NCE (n = 10)*		*CE (n = 18)*		*NCE (n = 18)*		*CE (n = 28)*		*Exposure*	*Gender*	*Exposure × Gender*
Total Speech	*Mean*	SD	*Mean*	SD	*Mean*	SD	*Mean*	SD	*(F)*	*(F)*	*(F)*
Communicators	46.1	20.3	51.2	24.5	25.5	12.4	41.8	21.1	0.105	9.31**	0.575
Nouns	16.7	11.3	19.2	10.7	29.4	12.3	21.2	14.1	1.63	4.83*	1.85
Verbs	14.4	7.8	11.8	9.2	12.5	7.7	11.3	6.6	0.024	0.332	0.395
Functors	17.9	11.1	14.3	10.7	27.3	9.7	20.6	10.6	4.00*	10.75**	0.390
Modifiers	4.8	4.3	3.5	3.2	5.2	5.1	5.1	6.4	0.020	1.02	0.255

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

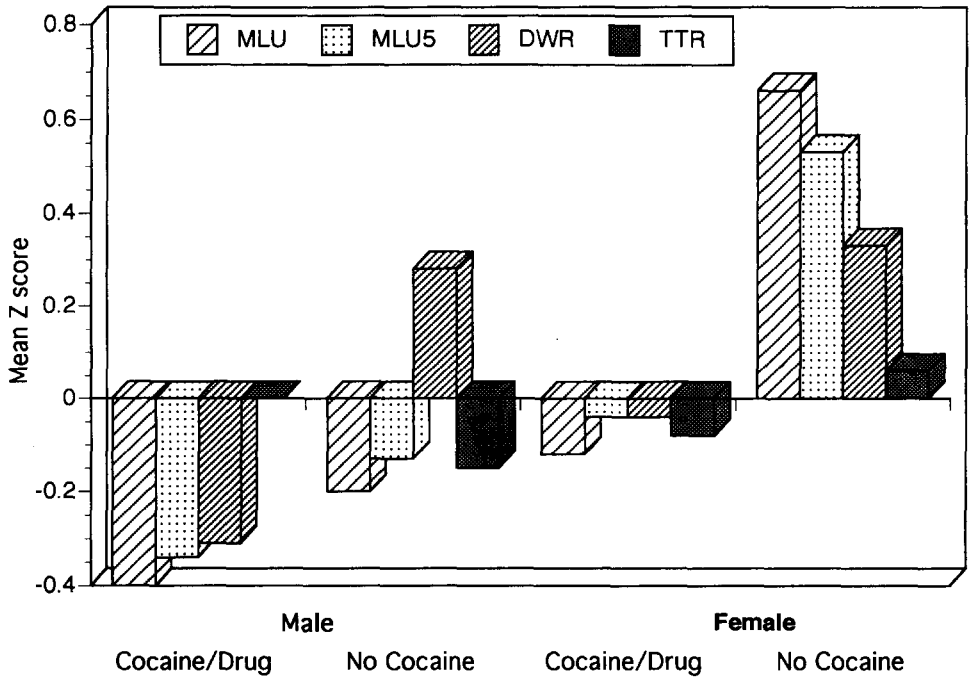

Figure 1. Mean Normalized (z) Scores for Child Mean Length of Utterance (MLU), Mean of 5 Longest Utterances (MLU5), Number of Different Word Roots (DWR), and Type-Token Ratio (TTR) by Drug-Exposure and Gender.

language patterns, while the cocaine-exposed girls show scores similar to both cocaine-exposed and non-cocaine-exposed boys.

Composite Language Score. Figure 1, using normalized scores, shows the consistent pattern in performance across the four scores. To capture this pattern statistically, a composite language measure was computed from the z-scores for four variables reflecting grammatical and lexical diversity: MLU, MLU5, TTR, and *number of different word roots.* Although closely related, MLU and MLU5 were both included as measures of grammatical complexity, as MLU5 provides additional information by reflecting the upper range of language skills. The low correlation between *number of different word roots* and TTR ($r = -.28$) underscores the differences between these two measures, and supports inclusion of both.

An ANCOVA revealed a significant covariate effect for *marijuana* ($F(1, 61) = 4.17, p < .05$). Non-cocaine-exposed toddlers showed a higher positive mean score ($M = .94$; $SD = 2.73$) than did cocaine-exposed toddlers ($M = -.58$, $SD = 2.18$), this difference achieving marginal significance ($F(1, 61) = 3.94, p < .06$). Gender emerged as a significant main effect ($F(1, 61) = 4.84, p < .05$), with girls showing greater complexity ($M = .45$, $SD = 2.4$) than did boys ($M = -.75$, $SD = 2.5$).

Caregiver Language

Table 5 presents the means and standard deviations for the measures of caregiver language. Outcome variables were entered in a two-way analysis of variance (ANOVA) with *exposure* and *child's gender* as between-group variables. There were no significant main effects of *exposure* for any measure of caregiver language. Both the grammatical complexity and the lexical diversity of the caregiver language directed toward girls and boys were also similar, with the exception of a marginally significant main effect of *gender* on *number of different word roots* ($F(1, 70) = 2.76$, $p < .10$). Caregivers produced a somewhat greater rate of different word types when the dyadic partner was a girl ($M = 21.0$, $SD = 3.8$) than when the partner was a boy ($M = 19.5$, $SD = 4.2$).

Measures of grammatical diversity showed a significant main effect for *gender* on *percent verbs* ($F(1, 70) = 5.41$, $p < .03$) and a marginally significant main effect on *percent functors* ($F(1, 70) = 3.41$, $p < .07$). Verbs comprised a significantly greater percent of the speech in interactions with boys ($M = 29.4\%$, $SD = 3.3$) than with girls ($M = 27.1\%$, $SD = 4.8$), whereas functors comprised a significantly greater percent of the speech in interactions with girls ($M = 38.0\%$, $SD = 5.5$) than with boys ($M = 35.6\%$, $SD = 4.9$). There were also marginally significant interaction effects between *gender* and *exposure* for *percent functors* ($F(1, 70) = 2.95$, $p < .10$). Functors comprised a similar proportion of the speech the non-cocaine using caregivers produced with both girls ($M = 37.3\%$, $SD = 5.9$) and boys ($M = 37.8\%$, $SD = 4.2$); caregivers in the cocaine-and-other-drug-exposed dyads used a smaller percentage of functors with boys ($M = 34.4\%$, $SD = 4.9$) than with girls ($M = 38.3\%$, $SD = 5.3$).

Discussion

Our primary focus in this study was to compare language development of prenatally cocaine-exposed toddlers with a cohort of their non-cocaine-exposed inner-city peers. Of primary concern was the developmental impact of the prenatal and postnatal environment associated with prenatal cocaine exposure, over and above that of poverty and its associated environmental stressors. In this sample of inner-city children, there is evidence for some delays in language development, regardless of exposure, when compared with reports from other investigators (Lawrence, 1984; Fenson et al., 1994). Our findings also suggest that children exposed to cocaine in utero and raised by a cocaine-using mother produce less complex language at 24 months than do their non-cocaine-exposed peers even after controlling for relevant perinatal outcome variables and other maternal drug use. These results further suggest that the effect of cocaine-exposure is qualified by gender, and is stronger for girls than for boys.

The present findings suggest that both cohorts, cocaine-exposed and non-exposed, demonstrate the language delays associated with poverty and disadvantaged environments. However, when comparing cocaine-exposed youngsters to their non-cocaine-exposed peers, a pattern of findings emerged, with non-cocaine-

Table 5. Mean and *SD* for Caregiver Grammatical Complexity, Lexical Diversity, and Composite Language Score by Gender and Exposure Status of the Child in the Dyad

	Boys				*Girls*						
	NCE (n = *10*)		*CE* (n = *18*)		*NCE* (n = *18*)		*CE* (n = *28*)		*Exposure*	*Gender*	*Exposure* × *Gender*
Measure	*Mean*	SD	*Mean*	SD	*Mean*	SD	*Mean*	SD	*(F)*	*(F)*	*(F)*
Rate of speech	62.99	11.54	62.19	11.22	64.20	17.38	69.93	16.96	0.637	1.64	0.574
MLU	3.19	0.49	2.93	0.55	3.19	0.46	3.13	0.46	1.76	1.27	0.284
Mean words/utterance	2.98	0.39	2.72	0.48	2.99	0.59	2.90	0.44	1.71	1.11	0.433
MLU5	9.22	1.77	9.08	2.23	9.31	1.90	9.21	1.40	0.069	0.077	0.003
Different word roots	20.41	4.37	18.94	4.21	20.07	3.76	21.63	3.71	0.211	2.76°	2.40
TTR	0.37	0.04	0.33	0.08	0.33	0.05	0.32	0.05	0.100	0.090	0.029
Composite language score[a]	0.35	2.53	−0.61	2.74	0.40	2.43	0.41	2.01	0.369	1.34	0.664

[a] Composite language score was calculated by summing the *z*-scores for MLU, MLU5, number different word roots, and TTR.
° $p < .10$.

exposed children producing more complex and more varied language. Non-exposed toddlers produced longer utterances, reflected a more complex grammatical structure, and showed a generally more complex level of language production (as revealed by the Composite Language Score).

Gender differences also qualified these findings, with girls showing more complex language production than did boys. The pattern of mean scores across the language variables, however, suggests that cocaine-exposed girls showed language skills similar to those of the boys, indicating the possibility of a developmental lag. Girls tend to acquire language at a slightly more rapid pace than do boys (Fenson et al., 1994; Huttenlocher et al., 1991; Morisset et al., 1995), and these findings may reflect a floor effect among the boys. That is, the boys in our sample may not have developed enough language skills to show a differential effect for exposure. If this is the case, further research with older children should reveal an effect of exposure for boys as well.

The findings of a greater effect of cocaine exposure on language development among girls than boys stands in contrast to earlier findings of Morisset et al. (1995), who found that in an economically disadvantaged population, language development in boys, as compared to girls, showed greater vulnerability to social stresses. It may be that the social stresses associated with an inner-city life-style depress language development in both the drug-exposed and non-exposed boys, thereby masking more subtle effects associated with prenatal and postnatal maternal drug use.

No clear differences associated with exposure grouping emerged in the linguistic complexity of caregiver input related to exposure status. No significant differences existed in the amount of grammatical complexity or lexical diversity of the caregivers' speech between the cocaine-and other-drug-using and the non-cocaine-using caregivers. The only significant difference among caregivers was in the effect of child's gender on grammatical diversity. These gender differences may be reflective of a more directive style with boys and a more conversational style with girls or could reflect differences in the selection of toys chosen by or for the different genders. The interaction effects may also reflect difference in the way caregivers in the two exposure groups interact with their children. Clearly, further research on the interaction style in dyadic play is necessary for a better understanding of the differences revealed in children's and caregivers' language.

The inability to address fully the effects of differential drug use in the postnatal environment because of continued drug use among participating women is an important limitation of this study. The women in the study were recruited on the basis of a history of prenatal cocaine use, and very few of them decreased or ended their cocaine and other drug use following the birth of the participating child. Thus, an insufficient number of women decreased their drug use to permit comparisons in language function between the cocaine-exposed children of mothers who decreased their drug use and those who continued to use drugs.

A number of factors associated with drug use might influence parenting style and be crucial to the differences in language development. Cocaine-use may effect neuropsychological functions underlying memory, attention, persistence, and task orientation (Ardila, Rosselli, & Strumwasser, 1991; Beery, Van, Herzberg, & Hin-

kin, 1993; Manschreck et al., 1990; O'Malley, Adams, Heaton, & Garwin, 1992), which may, in turn, influence the capacity to attend to or sustain an interaction with a child. Parents who are cocaine intoxicated may exhibit altered and non-normal patterns of interactions with their children, thereby creating or furthering a chaotic parenting style.

Impairments of maternal–child interactions, including diminished maternal responsiveness and reciprocity (Burns et al., 1991), problems structuring and mediating the environment (see Mayes, 1995), and disorganized attachment patterns or disturbed maternal–child relationships (Rodning, Beckwith, & Howard, 1991) have been reported for cocaine- and polydrug-using mothers. Differences in arousal regulation found in cocaine-exposed infants as compared to non-cocaine-exposed infants (Mayes et al., 1996) may further contribute to the impaired interaction. These findings contrast with the parental interaction behaviors that have been found to contribute to developmental competencies in the infant years: attention-organizing activities, initiating and responding to infant bids, emotional exchanges, and elaborating on infant communications (e.g., Barnard & Martell, 1995; Bornstein, 1995).

Importantly, a broad range of language skills is found within the sample of cocaine-exposed toddlers, as well as their non-exposed peers, reflecting, in part, heterogeneity in the pace of language development typical of the toddler years. Although the majority of the cocaine-exposed toddlers performed more poorly than did their non-cocaine-exposed peers, the full range of outcomes was similar for the two groups. Heterogeneity of outcome has been reflected in studies of prenatal cocaine exposure (Bender et al., 1995; Griffith et al., 1994; Malakoff et al., 1994), underscoring the complexity of the relationship between exposure and outcome. This heterogeneity found among our exposed sample is likely to be influenced by the duration and rate of drug use following the birth of the child and the degree of postnatal environmental chaos as well as by prenatal exposure.

The findings from this study confirm that prenatal cocaine exposure is an indicator that a child is at risk for language delays, a risk that may be greater than that due to being born into an inner-city environment. Exposure to crack cocaine in utero is a marker for a set of co-occurring risk factors that begin with pregnancy and continue into the all-important first years of life. These risk factors include both the direct biological effects of the drug exposure and the psychological and social disruptions associated with cocaine and other drug use.

In addition to the biological risk factors associated with prenatal drug exposure, neighborhood and familial poverty and violence, low educational attainment, violence, and chaotic parenting place many drug-exposed children in double jeopardy. The findings of the present study do not allow assessment of whether the cocaine exposure, per se, is directly responsible for negative developmental outcomes. Furthermore, these findings do not attribute the vulnerability to specific (or any) biological effects of cocaine on the "language-centers" of the brain during prenatal development. It is possible that prenatal cocaine exposure affects specific neuropsychological functions in both parent and child (e.g., regulation of attention and behavioral status), which in turn impair later-developing functions and skills. Additional longitudinal research is needed to understand the individual and combined

effects of environmental factors associated with both maternal cocaine use and inner-city poverty on early language development.

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NOTE

1. The Likelihood Ratio (G^2) is used owing to the number of cells with $n < 5$.

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