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Four-year language outcomes of children exposed to cocaine in utero

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Abstract

A large cohort of children exposed to cocaine in utero ($n = 189$) were followed prospectively from birth to 4 years of age and compared to nonexposed children ($n = 185$) on the Clinical Evaluation of Language Fundamentals—Preschool (CELF-P), a measure of receptive and expressive language abilities. Children exposed to cocaine in utero performed more poorly on the expressive and total language measures than nonexposed children after controlling for confounding variables, including prenatal exposure to alcohol, marijuana, and tobacco, as well as medical and sociodemographic variables. Children exposed to cocaine had more mild receptive language delays than nonexposed children and were less likely to have higher expressive abilities. Also, maternal factors such as language ability, performance IQ, race, and education correlated with child language abilities. Prenatal cigarette and marijuana exposure were related to deficits in specific language skills. Children placed in adoptive or foster care who were cocaine exposed demonstrated superior language skills compared to children exposed to cocaine who remained in biological relative or mother's care. These findings support a cocaine-specific effect on language skills in early childhood that may be modified with an enriched environment.

Keywords: Cocaine exposed; Language; Adoptive/foster care

1. Introduction

Recent studies have reported a variety of speech and language delays in children exposed to cocaine in utero when compared to matched nonexposed children [3,8]. The nature of these observed deficits has in part been dependent on the age of the children examined, the measures used to assess language development, and the methods used to establish cocaine exposure. Studies have also differed in their consideration of confounding factors such as multiple drug use, including exposure to cigarette smoking, alcohol use, and other medical/sociodemographic variables. As such, confounding variables have limited our efforts at making definitive statements about the impact of cocaine exposure on young children's language development. In addition, only a few studies have reported the placement of children in adoptive/foster care, and no study to date has reported outcomes for cocaine-exposed children based on the placement of the child in biological/family care versus foster/adoptive care. See Table 1 for a summary of these studies.

Early language findings from our laboratory at 1 year of age demonstrated that the amount of cocaine exposure inversely influenced language outcomes [25]. Infants who were heavily exposed to cocaine had lower auditory comprehension scores than nonexposed infants and lower total language scores than infants with lighter or no exposure. Using the total language score, more heavily exposed infants were also more likely to be classified as mildly delayed than were nonexposed infants [25].

Additionally, at 2 years of age, studies of children exposed to cocaine have demonstrated persistent language delays [1]. Two-year-olds with fetal exposure to cocaine were more delayed in semantic development than a

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Table 1
Summary of studies of children exposed to cocaine that reported speech/language testing

Study	Subjects	Measures	Prospective	Masked tester	Biological or adoptive foster care	Controlled for multiple drug use	Findings
Bandstra et al. [3]	236 exposed, 207 nonexposed	CELF-P, Nepsy Language Domain	Yes	Yes	Not reported	Yes	Cocaine effect on language
Singer et al. [25]	66 children with heavy cocaine exposure, 68 with lighter cocaine exposure, 131 nonexposed children	PLS-3	Yes	Yes	Not reported	Yes	More heavily exposed infants had lower auditory comprehension scores than nonexposed children
Delaney-Black et al. [8]	204 exposed, 254 nonexposed	AAPS, Language sample analysis	Yes		Not reported	Yes	Low language children more likely to be cocaine exposed
Kilbride et al. [18]	118 cocaine-exposed children, 41 nonexposed children	SICD	Yes	No	Yes	No	Case-managed children score higher than children who received routine care
Bland-Stewart et al. [5]	11 cocaine exposed, 11 nonexposed	SICD-R, Language sample analysis	Yes	Yes	Yes	No	Delays in semantic representation
Madison et al. [19]	25 exposed, 25 nonexposed	Speech sample, HOME	No	No	Not reported	No	Higher use of phonological processes in cocaine-exposed group
Hurt et al. [16]	76 exposed, 81 nonexposed	PLS-3	Yes	Yes	Not reported	No	No differences in receptive, expressive, or total language
Hawley et al. [14]	20 cocaine-exposed children, 24 nonexposed children	AAPS, TACL-R	No	No	Yes	No	No differences between cocaine-exposed and nonexposed children
Mentis and Lundgren [22]	5 exposed, 5 nonexposed	Language sample	No	No	1 participant in foster care	No	Pragmatic and syntax differences
Nulman et al. [23]	23 exposed, 23 nonexposed	Bayley Scales, McCarthy Scale, Reynell Scale	No	Yes	Yes	No	Lower language comprehension and expression
Angelilli, et al. [1]	29 language delayed, 71 control children	Record review	No	No	Not reported	No	More reported cocaine use during pregnancy in language delayed sample
Malakoff et al. [21]	21 children of cocaine-using mothers	PPVT, EOWPVT, SICD-R	No	No	Not reported	No	60% of children show language delay with more receptive problems than expressive

Abbreviations of tests: CELF-P = Clinical Evaluation of Language Fundamentals – Preschool; AAPS = Arizona Articulation Proficiency Scale; PPVT = Peabody Picture Vocabulary Test; EOWPVT = Expressive One Word Picture Vocabulary Test; SICD-R = Sequenced Inventory of Communication Development—Revised; PLS-3 = Preschool Language Scale—3rd Edition; TACL-R = Test of Auditory Comprehension of Language—Revised.

comparable group of nonexposed children [5]. Similarly, spontaneous language samples revealed delays in syntax, discourse, and pragmatics of language [22]. Phonological processing problems appear equivocal based on current research. One study reported increased use of phonological processes in 22- to 51-month-old children exposed to cocaine in utero [19]. While these studies suggest language delays across all domains at 2 years of age, a recent large, prospective, well-controlled study reported no significant differences between a cohort of cocaine-exposed children and control children at age 2 1/2 on several domains of language functioning [16].

Studies of cocaine-exposed preschool children at 4–6 years of age have also been equivocal with one study reporting receptive but not expressive language delays [4] and another study reporting expressive language delays [8]. Yet another study reported both receptive and expressive language delays [23]. A longitudinal research team followed children to 3, 5, and 7 years of age and found a stable cocaine-specific effect on total language functioning from 3 to 7 years [3].

Fetal cocaine exposure may be associated with language delays due to direct effects of drug exposure or to environmental risk factors associated with maternal drug use. Reports on the biological effects of prenatal cocaine exposure suggest that several mechanisms may adversely affect language development. First, cocaine exposure appears to affect prenatal neurotransmitter development [20,33] that may in turn affect the speed of processing of auditory information. Deficits in auditory speed of processing have been associated with language delays in nonexposed children [30]. Second, cocaine exposure may also disrupt arousal and attentional systems, which may result in poor auditory discrimination, attention, and

memory—all cognitive processes seen as essential components of language development. Third, reduced blood flow to the cerebral cortex has also been associated with prenatal cocaine exposure [30,37] and may result in poor information processing. Also, language deficits may be a function of the global cognitive deficits in our cocaine-exposed children over the first 2 years of life [26].

Despite the demonstrated impact of teratologic agents on language development, the impact of the postnatal environment on language development may be even greater than the biological effects of cocaine. To date however, caregiver effects on the language development of cocaine-exposed children have been less well documented. A cocaine-using mother may not provide an optimal language learning environment for her child and may be unable to initiate and sustain affective, social, and linguistic interactions critical for normal language development [23].

In contrast to non-drug-using women, drug- and alcohol-using women are more likely to have comorbid mood or personality disorders that interfere with parenting interactions typically designed to facilitate the development of communication skills [27,30]. Also, the impact of prenatal cocaine exposure may be overshadowed by the impact of an urban, low socioeconomic status environment [18].

The present study documents language abilities of children at 4 years of age who have been exposed to cocaine in utero. Four years of age roughly coincides with the conclusion of the period of basic language acquisition [29]. At 4 years of age, children lacking in basic language skills, including semantics, morphology, syntax, and memory, can be reliably assessed on measures with normative standards [36]. This study controls for multiple drug confounders, as well as demographic and psychosocial variables known to relate to child language development. Caregiver effects on language development were examined through comparisons of children in biological relative care to those children in adoptive/foster care.

2. Methods

2.1. Subjects

A total of 374 four-year-old children (189 cocaine exposed and 185 nonexposed) were seen at 4 years of age. The sample was drawn from a cohort recruited at birth from a large, urban, county teaching hospital to participate in a longitudinal study of the sequelae of fetal drug exposure. Women who were considered to be at high risk for drug use due to lack of prenatal care, behavior suggesting intoxication, a history of involvement with the Department of Human Services in previous pregnancies, or self-admitted drug use, were given drug toxicology screening. Urine samples were obtained immediately before or after labor and delivery and analyzed for the presence of cocaine metabolites (benzoylecgonine; BZE), cannabinoids (THC), opiates, PCP, and amphetamines. The Syva Emit method (Syva, Palo Alto, CA) was used for urine analysis. The specificity for BZE was 99% at a concentration of 0.3 mg/ml. Follow-up thin layer chromatography or gas chromatography analyses were performed.

Infants also had meconium drug analyses performed for cocaine and its metabolites, i.e., BZE, meta-hydroxybenzoylecgonine (m-OH-bze), cocaethylene, THC, opiates, PCP, amphetamines, and benzodiazepines [17]. Meconium specimens were collected from the newborn's diaper in the hospital by a nurse trained in the research protocol. When available, samples were accumulated over multiple diapers from the same infant, and no attempt was made to prevent contamination with urine. After collection, specimens were stirred for 5 min to insure homogeneity and stored in a refrigerated container. Further details concerning collection of meconium can be found in a separate report [2].

Screening assays were conducted using Abbott Diagnostics polarization immunoassay reagents (FPIA). Cutoff levels for drugs of interest were cocaine and metabolites: 25 ng/g; opiates: 25 ng/g; amphetamines: 100 ng/g; phencyclidine: 25 ng/g; and tetrahydrocannabinol: 25 ng/g. Confirmatory assays were conducted using gas chromatography-mass spectrometry (GC/MS) operated in electron impact, selected ion monitoring mode.

Cocaine-exposed infants were identified based on either positive infant meconium, maternal urine, or maternal self-report, while control infants were negative on all indicators. Women who used alcohol, marijuana, or tobacco during pregnancy were included in both groups. Cocaine-positive infants were subdivided into heavier and lighter categories, with classification determined by meconium screen indication of use greater than the 70th percentile or self-report greater than the 70th percentile for the users. A previous report [2] on the concordance of meconium concentration and maternal self-report measures of heavier versus lighter use in our sample indicated reasonable concordance between biological and self-report measures (correlations ranging from .32 to .57 between maternal report of the severity of cocaine use and the amount of cocaine, cocaethylene, BZE, and m-OH-bze detected in the offsprings' meconium). For 10 women from the entire sample who denied cocaine use, but whose infants' meconium screens were positive, self-report data were estimated by assigning the median score for the group (heavier/lighter) to which they were assigned based on meconium status.

The sample size for the original cohort was 415. From birth to 4 years, there were 11 deaths in this sample, 8 cocaine positive and 3 negative ($\chi^2 = 1.9$, $P < .17$). At 4 years of age, 30 (20 cocaine positive) of the surviving

subjects ($N = 404$) failed to have the language test. Of these children, 12 (8 cocaine positive) did not come to the visit, 16 (12 cocaine positive) dropped out, and 2 (1 cocaine positive) had moved out of state. Attrition was greater for the cocaine group ($P = .04$) compared with the control group. From enrollment at birth, the retention rate was 93% (374) for living children at 4 years. Of those 30 children who did not have test results at 4 years, 21 were cocaine positive. The 21 cocaine-positive children who did not participate were more likely to be White, with lower Hobel risk scores at birth, and to have mothers with lower Picture Completion scores than the remaining 189 cocaine-positive participants. The nine cocaine-negative children who did not participate had higher alcohol and cigarette exposure prenatally, higher gestational age, better Apgar score (5 min), and lower Hobel risk scores than the remaining 185 cocaine-negative participants.

2.2. Procedures

At 4 years of age, all children were administered the Clinical Evaluation of Language Fundamentals—Preschool (CELF-P) [36] and the prorated Wechsler Preschool and Primary Scales of Intelligence—Revised (WPPSI-R) [35] as part of their follow-up at the child development laboratory. The CELF-P is a standardized, normative language assessment comprised of Receptive and Expressive language subscales and includes measures of linguistic concepts, basic concepts, sentence structure, recalling sentences, formulating labels, and word structure. A summary score, the Total Language score, was also computed. All examiners were unaware of infant cocaine status.

To assess prenatal drug exposure, infants and their biologic mothers were seen immediately after birth, at which time the biologic mother was interviewed regarding drug use. Biologic mothers were asked to recall the frequency and amount of drug use for the month prior to pregnancy and each trimester of her pregnancy. More specifically, for tobacco, the number of cigarettes smoked per day was recorded; for marijuana, the number of joints smoked per day; and for alcohol, the number of drinks of beer, wine, or hard liquor per day was computed, with each drink equivalent to 0.5 oz of absolute alcohol. For cocaine, the number of rocks consumed and the amount of money spent per day were noted. For each drug, the frequency of use was recorded on a Likert scale ranging from 0 = *not at all* to 7 = *daily use*, with scores converted to reflect the average number of days per week a drug was used. The frequency of use was multiplied by the amount used per day to compute a severity of use score for the month prior to pregnancy and for each trimester. These scores were then averaged to yield a total score for prenatal exposure for each drug. This drug assessment was updated at each follow-up visit to provide a similar measure of current drug use, with the assessments also administered to the foster or relative caregiver to provide a measure of postnatal exposure for children placed out of maternal care.

Birth, demographic, and medical characteristics were taken from hospital records and included maternal race, age, parity, number of prenatal care visits, type of medical insurance, infant Apgar scores, and infant birthweight, length, and head circumference. At the enrollment visit, maternal socioeconomic status [15] and educational level were calculated. Maternal vocabulary score was measured using the Peabody Picture Vocabulary Test—Revised (PPVT-R) [10] and two subtests of the Wechsler Adult Intelligence Scale—Revised (WAIS-R) [34]. The Block Design (BD) and Picture Completion (PC) subtests from the WAIS-R enabled an estimate of nonverbal intelligence. The Brief Symptom Inventory (BSI) [9] is a standardized self-report scale that was also administered at birth and at the 4-year visit to obtain a measure of severity of psychological distress. The BSI yields a summary score, the General Severity Index (GSI), which is an indicator of overall stress symptoms. The Hobel Neonatal Risk Index [14] was computed to obtain a measure of neonatal medical complications. Also at the 4-year visit, the child's placement (either biologic mother/relative or foster/adoptive caregiver) was noted and the data on the current caregiver were updated to provide concurrent measures of caregiver psychological distress and drug use, including tobacco, alcohol, marijuana, and cocaine or other drug use. If the child had been placed with a new caregiver, intellectual measures were also updated. The Home Observation of the Environment (HOME)—Preschool version was administered to the caregiver in an interview format as a measure of the quality of the caregiving environment [6].

All children and mothers received transportation and a US$50 stipend for participation. The Institutional Review Boards of the participating hospitals approved the study and informed written consent was obtained from caregivers. All mothers were protected by a Writ of Confidentiality (DA-98-91) issued by the Department of Health and Human Services for this study, which prevented the principal investigator from being forced to release any research data in which subjects are identified even under court order or subpoena.

2.3. Statistical analysis strategy

Cocaine-negative and -positive mothers and children were compared on demographic variables, frequency and severity of drug use, and infant birth outcomes, as well as on language outcome variables, using t tests for continuous data and chi-square analyses for categorical variables. All positively skewed data including drug self-report measures, the GSI, and meconium quantification variables were normalized by log $(x + 1)$ transformations. Means and standard deviations are reported in terms of

the original distribution, with the transformations used in analyses.

Analysis of variance (ANOVA) was performed on the unadjusted CELF-P scores to assess overall group differences and differences by light versus heavy exposure, followed by hierarchical linear regression analysis to assess specific drug effects. Since the groups were not matched on chronological age, comparisons were made to determine the impact of this variable. No group differences in chronological age were observed with the cocaine positive group's mean age 4.08 (S.D. = 0.20, range 3.01–5.09) and non-cocaine group's mean age 4.08 (S.D. = 0.19, range 3.9–5.09). Since the groups did not differ in chronological age, it was eliminated as a covariate.

Regression models designed to predict adjusted CELF-P scores retained confounding variables demonstrated previously to be significant in the regression model, as well as group status (cocaine exposed and nonexposed). Follow-up analyses using logistic regression controlling for covariates were completed on subscales with group differences (P=.20 or less).

In an effort to clarify the nature of the regression analyses, the following strategy for variable entry was employed. First, Pearson product moment or Spearman rank order correlations were used to assess the relationships among prenatal drug exposure and the demographic and medical factors to language outcomes. Confounding variables were entered stepwise into the hierarchical linear model if they were correlated with the outcome at $P<.20$ and were significantly different between the exposed and unexposed groups at $P<.05$. If, upon entry, covariates were significant at $P<.10$, they remained in the model. The order of entry was designed to account for demographic, environmental, and medical factors before drug exposure factors, consistent with a teratologic model [11,33] and to reduce the number of correlated variables in the statistical model. Demographic and prenatal factors were considered first, followed by environmental variables and drug exposure variables in the following order: HOME scale, maternal age, parity, number of prenatal care visits, maternal years of education, marital status, socioeconomic status, biological and current caregiver PPVT-R, WAIS-R BD, and PC scores biological and current caregiver psychological distress, and prenatal and current caregiver measures of cigarette, alcohol, and marijuana exposure, prior to cocaine exposure that was entered on the last step. Infant birth parameters, which can also be affected by cocaine use, were considered potentially mediating variables if they were related to the language outcome and different by cocaine group. Specifically, birthweight, length, head circumference, and gestational age were assessed by entering them into the model after all other variables if a significant cocaine effect emerged. Effects of PIQ were also considered for language variables predicted by cocaine use. Child's race and sex, which did not differ between the exposed and nonexposed groups, were considered moderator variables and their effects were tested through interaction terms. All analyses were performed using SAS 8.2 (SAS Institute Inc., Cary, N.C.).

Individual difference analyses were conducted using chi-square analyses to assess the likelihood that cocaine effects influenced receptive, expressive, and total language scores in the range of mild (standard scores below 80) and moderate delay (standard scores below 70), as well as for those children above the normative mean (standard scores above 100). Finally, cocaine-exposed children in foster/adoptive care and biologic maternal or relative care and nonexposed children were also compared on a CELF-P summary and subscale outcomes using ANOVA. In the event of a significant group effect, follow-up pairwise t tests were performed using Tukey's method.

3. Results

3.1. Sample characteristics

Child and maternal characteristics of the sample are presented in Tables 2 and 3. Cocaine-using women and controls were primarily African American of low-income and single marital status. The cocaine-using women were older, had more children, and received fewer prenatal care visits than nonusers. In addition, cocaine-using women used other drugs more frequently and in higher amounts than nonusers. Of cocaine users, 66 (49%) were classified as heavier and 68 (51%) as lighter users.

Table 2
Demographics of cocaine-exposed and nonexposed children

	Cocaine exposed (n=189), M (S.D.)	Nonexposed (n=185), M (S.D.)	$\chi^2/t/F$
Birthweight (g)[+]	2698 (647)	3100 (697)	38.3****
Gestational age (weeks)	37.7 (2.8)	38.4 (2.8)	2.40**
Birth length (cm)[+]	47.2 (4.0)	49.1 (3.7)	18.8****
Head circumference (cm)[+]	32.3 (2.1)	33.4 (2.4)	26.5****
Apgar, 1 min	8.01 (1.4)	7.92 (1.7)	−.57
Apgar, 5 min	8.78 (0.7)	8.78 (0.7)	−.09
Hobel Risk Score	7.55 (16.6)	5.80 (15.7)	−1.05
African American, n (%)	156 (82.5)	148 (80.0)	.40
Female, n (%)	84 (44.4)	89 (48.1)	.51
Performance IQ (4 years)	84.8 (15)	88.1 (15)	2.13**
Verbal IQ (4 years)	79.8 (12)	82.1 (13)	1.80*
Full Scale IQ (4 years)	80.3 (13)	83.4 (14)	2.28**

* $P<.1$.
** $P<.05$.
**** $P<.001$.
[+] adjusted for gestational age.

Table 3
Maternal and current caregiver characteristics of cocaine-exposed and nonexposed children

	Mothers of cocaine exposed children, *M* (S.D.)	Mothers of nonexposed children, *M* (S.D.)	t/χ^2
Biological maternal			
Age at birth	29.7 (5.0)	25.6 (4.8)	−8.2****
Parity	3.5 (1.8)	2.7 (1.8)	−4.4****
Number of prenatal visits	5.1 (4.6)	8.8 (4.8)	7.6****
Prenatal care, *n* (%)	152 (80.4)	168 (90.8)	8.17***
Low socioeconomic status, *n* (%)	184 (97.9)	181 (97.8)	0.001
African American, *n* (%)	157 (83.1)	149 (80.5)	0.40
Married, *n* (%)	16 (8.5)	32 (17.3)	6.5***
Years of education	11.6 (1.7)	12.0 (1.4)	2.6***
Employed, *n* (%)	11 (5.9)	38 (20.7)	17.8****
Amount of drug use during pregnancy			
Tobacco [b]	11.6 (11.2)	4.1 (7.7)	−10.5****
Alcohol [c]	9.7 (17.6)	1.4 (4.6)	−10.6****
Marijuana [c]	1.4 (3.5)	0.6 (3.5)	−4.3****
Cocaine[c]	24.7 (46.1)	–	
PPVT-R score	73.6 (15.3)	77.8 (14.9)	2.7***
WAIS-R BD score	6.8 (2.1)	7.2 (2.1)	1.4
WAIS-R PC score	6.7 (2.1)	7.1 (2.4)	1.4
Global Severity Index	0.81 (0.7)	0.49 (0.5)	−5.2****
Current caregiver at 4 years[a]			
PPVT-R score	78.9 (17.5)	78.2 (15.6)	−0.39
WAIS-R BD score	6.8 (2.3)	7.2 (2.1)	1.59
WAIS-R PC score	6.9 (2.3)	7.0 (2.4)	0.41
Global Severity Index	0.32 (0.38)	0.38 (0.43)	1.3
Amount of drug use (past 30 days)			
Tobacco[b]	7.2 (8.3)	4.4 (7.5)	−4.1****
Alcohol[c]	2.4 (6.7)	2.1 (7.3)	−0.8
Marijuana[c]	0.15 (0.76)	0.42 (3.15)	0.87
Cocaine[c]	5.2 (45.6)	0	0.22**
HOME Scale at 4 years	42.0 (6.4)	41.7 (6.7)	−0.5

PPVT-R = Peabody Picture Vocabulary Test—Revised; WAIS-R BD = Wechsler Adult Intelligence Scale—Revised Block Design Score; WAIS-R PC = Wechsler Adult Intelligence Scale—Revised Picture Completion Score.

[a] Primary female caregiver to child at 4 years of age.

[b] Mean number of cigarettes per day.

[c] Mean number of drinks, joints, or "rocks"/day × mean number of days/week.

* $P<.1$.

** $P<.05$.

*** $P<.01$.

**** $P<.001$.

Lighter users averaged 6.4 ± 4 (ranging from 0.1 to 16.5) and heavier users 36.3 ± 40 (ranging from 0.1 to 175) units ("rocks") of cocaine per week over the pregnancy. Cocaine-exposed infants were more likely to be preterm and of lower birthweight, head circumference, and birth length, after adjustment for gestational age, than nonexposed infants.

At birth, 49 (26%) exposed infants were placed outside maternal/biologic care. Those children placed outside of maternal/biologic care were more likely to be more heavily exposed. In contrast, only 3 (2%) of nonexposed infants were placed outside of maternal/biologic care. By 4 years, 42 (22%) cocaine-exposed children were placed in adoptive/foster care, with equivalent distribution of children with lighter versus heavier exposure, compared to 10 (8%) nonexposed children.

3.2. Relationship of CELF-P outcomes to prenatal drug exposure and caregiver characteristics

The relationship of language outcomes to current caregiver characteristics and prenatal drug exposure is shown in Table 4. Maternal race was correlated with all three language scores and five out of six language subtest scores with African American mothers scoring more poorly than mothers who were not African American. Lower maternal and current caregiver vocabulary scores (PPVT-R) were related to poorer receptive, expressive, and total language scores. Lower maternal WAIS-R block design score was associated with lower receptive and total language scores.

3.3. Language outcomes

Language scores were adjusted for confounding variables shown to be significantly related to the language measures by regression analyses. After controlling for significant confounding variables, children who were cocaine exposed differed from children who were not exposed on the Expressive Language subscale score, as well as the Total Language score of the CELF-P. Analyses of the individual subtests of the CELF-P showed significant differences by group on the Basic Concepts subtest only (see Table 5). There were no gender interaction effects; however, PIQ significantly predicted language outcomes (see Appendix A). Heavier versus lighter exposed groups did not differ significantly on the receptive, expressive, or total language scores of the CELF-P. There were nonsignificant trend effects suggesting cocaine-exposed children tended to score lower on the Formulating Labels and Sentence Structure subtests than did nonexposed children.

Children were also classified as demonstrating mild (standard scores below 80) or moderate (standard scores below 70) language disorders and high language abilities (standard scores above 100). Group differences were observed in the number of children with mild disorders on the Receptive subscale and the number of children with high language abilities on the Expressive subscale (see Table 6), with cocaine-exposed children more apt to be represented in the mild disorders group than nonexposed children. Logistic regression analysis of high language abilities was conducted with and without

Table 4
Correlation of maternal, current caregiver characteristics, birth outcomes, and prenatal drug exposure with child language

Variable	Receptive language	Linguistic concepts	Basic concepts	Sentence structure	Expressive language	Recalling sentences	Formulating labels	Word structure	Total language
Biological maternal									
Race	−.24****	−.19****	−.26****	−.17****	−.13**	.00	−.25****	−.12**	−.20****
PPVT-R	.10**	.12**	.13**	.05	.10*	.03	.21****	.11**	.11**
WAIS-R Block Design	.15***	.14***	.13**	.11**	.09*	.00	.18****	.06	.13**
GSI at Birth	−.03	−.03	−.10	−.01	−.01	−.03	.05	−.08	−.03
Prenatal Drug Exposure									
Cigarettes[b]	.03	.01	−.02	.11**	−.02	−.12**	.10*	−.01	.00
Alcohol[c]	−.03	.00	−.08	.01	−.08	−.13**	−.01	−.04	−.06
Marijuana[c]	−.00	.00	−.01	.01	.05	.01	.11**	.05	.02
Cocaine[c]	−.07	−.06	−.12**	.01	−.11**	−.15***	−.01	−.06	−.09*
Current caregiver at 4 years[a]									
Caregiver PPVT-R	−.18***	.09*	.19****	.16***	.14***	.01	.30****	.14***	.17***
Caregiver GSI	−.08	−.08	−.13**	−.04	−.04	−.05	.00	−.03	−.07
Amount of drug use (past 30 days)									
Cigarettes[b]	−.04	.05	−.07	−.07	−.03	−.06	.0*	−.01	−.03
Alcohol[c]	−.02	−.01	−.02	.05	−.03	−.06	−.02	.004	−.03
Marijuana[c]	−.12**	−.11**	−.05	−.13**	−.11**	−.10*	.10*	−.11**	−.12**
Cocaine[c]	−.04	−.06	.005	.06	−.05	−.04	−.02	−.05	−.04*
HOME Score at 4 years	.29****	.07	.18***	.20****	.31****	.18***	.28****	.19***	.32****
Infant birth outcomes									
Gestational age	.04	.09*	.04	−.02	.04	.03	.01	.04	.05
Birthweight	.05	.06	.09*	.003	.06	.06	.03	.06	.06
Birth length	.07	.08	.08	.03	.09*	.08	.07	.10**	.08*
Head circumference	.10*	.11**	.13**	.05	.10*	.09*	.07	.10*	.11**
ng/g[d] cocaine	−.02	.03	−.08	.00	−.04	−.09	.04	−.03	−.03
Ng/g[d] BZE	−0.3	.01	−.11**	.01	−.10*	−.13**	−.02	−.06	−.07
ng/g[d] THC	.03	.06	.01	.02	.05	.03	.08	.06	.05

[a] Primary female caregiver to child at 4 years of age.
[b] Mean number of cigarette per day.
[c] Mean number of drinks, joints, or "rocks"/day × mean number of days/week.
[d] Nanograms per gram of metabolite found in infant meconium.
* $P<.10$.
** $P<.05$.
*** $P<.01$.
**** $P<.001$.

Table 5
Adjusted language scores at 4 years

	Cocaine exposed ($n=189$), M (SE)	Nonexposed ($n=185$), M (SE)	F	P
Receptive language[a]	77.2 (1.13)	79.6 (1.14)	2.3	.13
Linguistic concepts[a]	5.5 (0.22)	5.8 (0.22)	0.8	.36
Basic concepts[b]	6.0 (0.21)	6.8 (0.21)	6.9	.01
Sentence structure[c]	6.3 (0.21)	6.8 (0.21)	2.9	.09
Expressive language[d]	82.1 (0.91)	85.4 (0.93)	5.3	.02
Recalling sentences[e]	7.4 (0.24)	8.0 (0.24)	2.6	.11
Formulating labels[f]	6.8 (0.18)	7.2 (0.18)	2.7	.10
Word structure[b]	6.7 (0.18)	7.0 (0.18)	0.9	.33
Total language[a]	79.3 (0.94)	82.1 (0.95)	4.4	.04

[a] Adjusted for HOME and maternal marital status at birth.
[b] Adjusted for HOME and parity.
[c] Adjusted for HOME and prenatal average cigarette exposure.
[d] Adjusted for HOME.
[e] Adjusted for HOME, prenatal average cigarette exposure, and prenatal average marijuana exposure.
[f] Adjusted for HOME, maternal marital status at birth, biologic maternal PPVT-R, prenatal average cigarette exposure, and prenatal average marijuana exposure.

Table 6
Comparisons of mild, moderate, and high language skills

Language skill	Cocaine exposed ($n=189$)	Nonexposed ($n=185$)	χ^2	P
Receptive mild disorder <80	108 (57.1%)	86 (46.5%)	4.25	.039
Receptive moderate disorder <70	60 (31.8%)	53 (28.7%)	0.43	.514
Receptive high abilities >100	15 (7.9%)	20 (10.8%)	0.91	.340
Expressive mild disorder <80	63 (33.3%)	56 (30.3%)	0.40	.525
Expressive moderate disorder <70	28 (14.8%)	28 (15.1%)	0.01	.931
Expressive high abilities >100	12 (6.4%)	26 (14.1%)	6.08	.014
Total mild disorder <80	93 (49.2%)	74 (40.0%)	3.20	.073
Total moderate disorder <70	47 (24.9%)	49 (26.5%)	0.13	.720
Total high abilities >100	11 (5.8%)	18 (9.7%)	1.99	.157

Table 7
Logistic regression on total language >100

	OR (95% CI)	*P* value
HOME score at 4 years	1.19 (1.08–1.31)	.0004
Biological maternal PPVT-R	1.03 (0.99–1.06)	.080
Prenatal average cigarette exposure per day	1.68 (1.17–2.42)	.005
Prenatal cocaine exposure	0.35 (0.13–0.92)	.030

controlling for PIQ. The HOME score, prenatal cigarette use, and prenatal cocaine use were significant predictors of language scores (see Table 7 and Appendix B).

3.4. Comparisons of maternal/biologic and adoptive/foster caregivers

Adoptive/foster mothers had higher verbal IQs and higher HOME scores than maternal/biologic caregivers. Cocaine-exposed children in adoptive or foster care performed better on the Basic Concept subtest of the CELF-P than cocaine-exposed children who remained in maternal or biological relative care. While the cocaine-exposed children in maternal/biological care performed more poorly than nonexposed children on the Total Language and Expressive Language scales, as well as on the Basic Concept and Recalling Sentences subtests, the cocaine-exposed children placed in adoptive/foster care did not differ from nonexposed children who were in less stimulating and lower social class homes (see Table 8).

4. Discussion

The results of this study confirm that children exposed to cocaine in utero continue to lag behind their nonexposed counterparts in language skills at 4 years of age. These deficits persist even when confounding factors are controlled. While the differences in language skills of children with heavier versus lighter cocaine exposure appear to vanish by 4 years of age, effects of adoptive/foster care placement on the outcomes of the more heavily exposed children emerge.

Previous reports comparing cocaine-exposed to nonexposed children at preschool have been equivocal regarding whether language differences between these groups are primarily receptive, expressive, or both [3,4,8,23]. Our findings suggest that deficits differ depending on whether comparisons include the entire cohort of children (with children falling both in the normal and disordered range) or only children exhibiting lower scores (<80 standard score) that classify them as having language disorders. When the groups are compared as a whole, these language differences are more pronounced in expressive than receptive abilities. However, when children with mild disorders were considered (<80 standard score), receptive deficits were more apparent than expressive. There is some evidence to suggest that patterns of language skills of children whose abilities fall at the extremes of the distribution of language skills (high or low) differ from the pattern of abilities observed in the cohort as a whole. The genetic or environmental contributions to individual differences in language ability at the extremes of the normal distribution can differ from those that influence language abilities of the rest of the population [24]. For example, Viding et al. [31] found that genetics influences on language impairment at 4 years of age increase at the low end of the distribution. In our study, we found that children at the low end of the distribution exhibited more deficits in receptive than expressive language, while children with high language skills, the high tail of the distribution, differed on expressive abilities.

Table 8
Bivariate comparisons of key characteristics and language outcomes by caregiver group

	Cocaine-exposed maternal biologic ($n=147$)	Cocaine-exposed adoptive/foster ($n=42$)	Nonexposed ($n=185$)	*F*	*P*
Prenatal cocaine exposure[a]	2.3 (1.2)	2.8 (1.4)	–	6.72	.010
Home score at 4 years[b,c]	41.0 (6.4)	45.5 (5.1)	41.7 (6.7)	8.01	.0004
Current caregiver PPVT-R[b,c]	76.4 (16.5)	89.1 (17.9)	78.2 (15.6)	8.29	.0003
Receptive language	76.0 (15.9)	80.8 (13.9)	79.7 (16.8)	2.67	.071
Linguistic concepts	5.6 (2.9)	5.1 (2.8)	5.9 (3.2)	1.21	.301
Basic concepts[b,d]	5.7 (2.7)	7.1 (3.2)	6.8 (3.0)	7.72	.001
Sentence structure	6.3 (2.7)	7.2 (2.6)	6.6 (2.8)	1.87	.156
Expressive language[d]	81.7 (12.8)	85.6 (10.8)	85.3 (14.0)	3.51	.031
Recalling sentences[d]	7.1 (2.8)	7.7 (2.6)	8.2 (3.1)	5.77	.003
Formulating labels	6.6 (2.4)	7.6 (2.1)	7.0 (2.5)	3.04	.049
Word structure	6.6 (2.5)	7.0 (2.4)	7.0 (2.6)	1.19	.307
Total language[d]	78.4 (13.0)	82.3 (11.4)	82.1 (14.5)	3.48	.032

[a] Mean number of "rocks"/day × mean number of days/week.
[b] Biococaine differs from adoptive/foster care.
[c] Adoptive/foster care differs from nonexposed.
[d] Biococaine differs from nonexposed.

Further examination of specific language skills revealed that not all aspects of language are deficient. Analyses of the subtests of the CELF-P revealed significant differences on the Basic Concepts subtest, but not Linguistic Concepts, Recalling Sentences, or Word Structure subtests. The Basic Concept subtest assessed the child's knowledge of attributes, dimension/size, direction, location, position, number, quantity, and equality. This subtest may be less linguistically based than other sub-tests and more influenced by cognitive development. Language scores were associated with PIQ, suggesting that language skills may be influenced by more general cognitive abilities. An enriched environment such as that provided by adoptive/foster placement may nurture these skills. The postnatal environment may have a greater impact on these language skills than fetal exposure to cocaine.

Although this study was not designed to assess the effects of cigarettes and marijuana, our findings support those of Fried and Watkinson [12] who noted poor language development after prenatal tobacco exposure in 3- and 4-year-old children. Two explanations for these language differences may be that cocaine exposure has an effect on reducing both auditory processing [21] and attentional abilities [13]. The negative effects of marijuana exposure on language in this sample appeared consistent with findings of a negative association of memory and verbal outcomes at 3 years in a Canadian study [12] and impairments in short-term memory and verbal reasoning in 3-year-old marijuana-exposed children in Pittsburgh [7].

In our cohort, children placed in adoptive/foster care were smaller and younger at birth and more heavily exposed to cocaine prenatally than exposed children who remained in maternal/biologic care. The fact that more heavily exposed children placed in foster care performed relatively better at age 4 may be due to a more stimulating environment for language development as evidenced by the superior HOME score and Verbal IQ score of the adoptive/foster mothers. Exposed children placed in adoptive/foster care performed similarly to nonexposed children on the language measures at 4 years, although nonexposed children had, by operationally defined measures, less optimal home environments with less educated and less verbal caregivers. While it is encouraging that an enriched environment may stimulate language skills, further research is needed to determine the specific characteristics of the environment that contribute to language development. It is not known to what extent the environment can improve language abilities and if these gains will persist into the school years. In sum, cocaine-specific effects on language skills persist at 4 years of age. However, language skills are influenced both by general cognitive abilities and environmental influences.

4.1. Strengths and limitations of the study

A major strength of this study is the large cohort, followed from birth, with cocaine exposure documented by both maternal and child biological markers collected perinatally, in addition to maternal report at the time of birth. Multiple drug confounders, as well as demographic and psychosocial variables known to relate to child language development, were controlled. A unique aspect of the study is the evaluation of caregiver effects on language as children in biological relative care were compared to those children in adoptive/foster care.

Potential limitations of the study include the use of a single standardized language measure, inaccurate reporting of maternal drug use throughout each trimester of the pregnancy, and the lack of a control group of nonexposed children placed in adoptive/foster care. Turning first to the language measure, the CELF-P may not have been sensitive enough to detect subtle language differences. Assessment of conversational language samples might have provided a more naturalistic assessment of language capabilities. Language skills not included in the assessment such as pragmatics and narrative abilities may be critical for understanding everyday language competence. Finally, the CELF-P may not be a sensitive assessment of language skills in a primarily low SES, African American population. Another limitation of the study is the use of maternal self-report of the quantity and trimester of cocaine use. A final limitation is the lack of a control group of children placed in adoptive/foster care who were not cocaine exposed. In our sample, few children in the control group were placed in adoptive/foster care. Thus, we do not know how children who were not cocaine exposed but in adoptive/foster care may have performed on the language measures.

Despite these limitations, our findings represent the most definitive statement on the prenatal cocaine effects on the development of language skills. Our estimate of cocaine effects was somewhat hindered by sample attrition, especially when the subjects lost were most impaired. Nonetheless, the fact that we find specific cocaine effects on language development strengthens our contention that prenatal cocaine has deleterious effects on language development.

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Appendix A. Regression analysis of language measures with PIQ

	Without PIQ		With PIQ	
	β (S.E.)	P value	β (S.E.)	P value
Receptive language				
HOME score	0.71 (0.12)	<.0001	–	–
Marital status at birth	4.62 (2.41)	0.056	–	–
Cocaine exposure	−2.44 (1.61)	0.130	–	–
Linguistic concepts				
HOME score	0.08 (0.02)	0.002	–	–
Marital status at birth	1.41 (0.47)	0.003	–	–
Cocaine exposure	−0.29 (0.31)	0.360	–	–
Basic concepts				
HOME score	0.11 (0.02)	<.0001	0.05 (0.02)	0.030
Parity	−0.10 (0.08)	0.220	−0.01 (0.07)	0.840
Cocaine exposure	−0.79 (0.30)	0.009	−0.56 (0.27)	0.040
Performance IQ	–	–	0.09 (0.01)	<.0001
Sentence structure				
HOME score	0.12 (0.02)	<.0001	–	–
Prenatal average cigarette exposure per day	0.32 (0.12)	0.010	–	–
Cocaine exposure	−0.55 (0.32)	0.090	–	–
Expressive language				
HOME score	0.65 (0.10)	<.0001	0.34 (0.09)	<.0001
Cocaine exposure	−2.99 (1.30)	0.020	−1.54 (1.17)	0.190
Performance IQ	–	–	0.42 (0.04)	<.0001
Recalling sentences				
HOME score	0.09 (0.02)	0.0002	–	–
Prenatal average cigarette exposure per day	−0.11 (0.14)	0.430	–	–
Prenatal average alcohol exposure per week	−0.16 (0.15)	0.310	–	–
Cocaine exposure	−0.59 (0.37)	.0110	–	–
Formulating labels				
HOME score	0.11 (0.02)	<.0001	–	–
Marital Status at birth	0.90 (0.36)	0.010	–	–
PPVT-R Standard Score	0.03 (0.01)	0.002	–	–
Prenatal average cigarette exposure per day	0.27 (0.11)	0.010	–	–
Prenatal average marijuana exposure per week	0.36 (0.19)	0.050	–	–
Cocaine exposure	−0.46 (0.28)	0.100	–	–
Word structure				
HOME score	0.10 (0.02)	<.0001	–	–
Parity	−0.12 (0.07)	0.070	–	–
Cocaine exposure	−0.25 (0.26)	0.330	–	–
Total language				
HOME score	0.67 (0.10)	<.0001	0.33 (0.09)	0.0004
Marital Status at birth	2.85 (2.01)	0.160	0.35 (1.76)	0.840
Cocaine exposure	−2.82 (1.34)	0.040	−1.44 (1.17)	0.220
Performance IQ	–	–	0.46 (0.04)	<.0001

Appendix B. Logistic regression on total language >100 with performance IQ

	No PIQ		With PIQ	
	OR (95% CI)	P value	OR (95% CI)	P value
HOME score at 4 years	1.19 (1.08–1.31)	.0004	1.16 (1.04–1.29)	.007
Biological maternal PPVT-R	1.03 (0.99–1.06)	.080	1.01 (0.98–1.04)	.460
Prenatal average cigarette exposure per day	1.68 (1.17–2.42)	.005	1.70 (1.14–1.23)	.010
Prenatal cocaine exposure	0.35 (0.13–0.92)	.030	0.42 (0.15–1.23)	.112
Performance IQ	–	–	1.07 (1.03–1.11)	.0001

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