Developing Language Skills of Cocaine-Exposed Infants

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ABSTRACT. *Objective.* **To assess whether there is an association of level of fetal cocaine exposure to developmental precursors of speech-language skills at 1 year of age, after controlling for confounding factors.**

Design. **In a prospective, longitudinal, quasi-experimental, matched cohort design, 3 cocaine exposure groups were defined by maternal self-report and infant meconium assay: nonexposure (n = 131), heavier exposure (n = 66), >the 75th percentile for maternal self-report and >the 70th percentile of benzoylecgonine concentration, and all others as lighter exposure (n = 68). At 1 year of age, the Preschool Language Scale-3 was administered by examiners unaware of infant drug status.**

Results. **Independent of confounding drug, medical, and environmental factors, more heavily exposed infants had lower auditory comprehension scores than nonexposed infants and lower total language scores than lighter and nonexposed infants. More heavily exposed infants were also more likely to be classified as mildly delayed by total language score than nonexposed infants. There were positive linear relationships between the concentration of benzoylecgonine in meconium and all outcomes and between maternal report of severity of prenatal cocaine use with poorer auditory comprehension indicating a relationship between amount of exposure and poorer outcomes.**

Conclusions. **This study documents significant behavioral teratogenic effects of fetal cocaine exposure on attentional abilities underlying auditory comprehension skills considered to be precursors of receptive language. Pediatricians are in a unique position to monitor early development of cocaine-exposed infants and make timely referrals for intervention.** *Pediatrics* **2001;107:1057–1064;** ***cocaine, language, infant development, drug exposure, attention.***

ABBREVIATIONS. BZE, benzoylecgonine; m-OH-bze, meta-hydroxylbenzoylecgonine; THC, cannabinoids; PCP, pentachlorophenol; PLS-3, Preschool Language Scale-3; GSI, Global Severity Index.

Since the epidemic of the 1980s, the number of infants exposed prenatally to cocaine, especially in poor, urban areas of the United States, has been of persistent concern. Cocaine is known to cross the placental barrier[1] and has primary effects on neurotransmitters implicated in fetal neuronal development,[2] as well as on uterine vascular flow.[3] Thus, its potential to negatively affect later learning abilities, including language, has been a subject of recent investigation.

Studies that have examined language outcomes to date report equivocal findings. Several studies have found cocaine-exposed, preschool children to perform more poorly than nonexposed children on various measures of language functioning.[4–11] Two-year-olds with fetal exposure to cocaine were found to be more delayed in semantic development than a comparable group of nonexposed children,[9] and 2- to 4-year-old children referred to a clinic for language delays were more likely to be cocaine-exposed than children with normal language development.[7] In an innovative study, 23 adopted, preschool, cocaine-exposed children were compared with children of similar social class and maternal IQ and found to have poorer verbal comprehension and expressive language.[11] Bender found receptive language delays on the Preschool Language Scale-3 (PLS-3) in 18 prenatally exposed 4- to 6-year-old children in comparison with 2 groups of children; those not prenatally exposed but whose mothers currently used cocaine and children without any history of exposure.[4] Similarly, a large, prospective study of children of cocaine-using mothers who received drug treatment during pregnancy found lower verbal reasoning skills on standardized tests at 3 years old compared with drug-free infants.[10]

However, in a controlled study of 20 exposed children and 24 nonexposed children at preschool age, few differences in language development were found.[12] Similarly, a recent large, prospective, well-controlled study reported no significant differences between a cohort of cocaine-exposed children and control children at age 2½ on several domains of language functioning.[13]

Methodologic difficulties of most previous studies preclude clear interpretation of their results. Many previous studies had small sample sizes,[4–9] retrospective case identification,[4,7,8,11] and tested children at various ages.[4,7,11,12] Frequently, confounding drug and caregiving factors known to be associated with maternal cocaine use, and also known to relate to child language abilities, were not controlled.[4,7,11] In the largest prospective studies,[10,13] attrition rates were high, with retention of only 50% to 72% of the cohort at follow-up. Without information on the children who were lost to follow-up, it is not possible to determine if correlates of attrition differed based on cocaine status, thus biasing the sample retained.

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Another issue in studies that have not found differences in language between cocaine-exposed and nonexposed children may have been the failure to consider severity of cocaine exposure, because several studies have now demonstrated that some behavioral deficits in cocaine-exposed populations may be detectable only at higher thresholds of exposure.[14–16] Also, few studies to date have used biological markers of degree of exposure, although these have been found to enhance reliability of classification and the ability to detect drug effects.[17] More reliable classification of exposure severity and status may be derived from the use of quantitative analyses of cocaine metabolite concentration in meconium,[17–19] which have not been used in studies of language outcomes to date.

The present study attempted to improve on previous studies by comparing infant behaviors underlying speech-language development in a large cohort of 1-year-old cocaine-exposed infants to that of controls, after consideration of exposure to multiple drug confounders, and demographic and psychosocial variables known to relate to child language development.[20] In particular, known correlates of child language outcome were considered,[21] including maternal nonverbal intelligence and vocabulary, educational level, psychological distress, and infant out-of-home placement. Maternal psychological distress is an important variable to assess in children's language outcomes because it has been found to be related to preschool cognition and to language outcomes in previous studies.[22–24] Drug- and alcohol-using women have been found to be more likely than nondrug-using women to have comorbid mood or personality disorders that interfere with parenting interactions that facilitate communicative development.[22,25,26]

METHODS

Participants

A total of 265 1-year-old infants (134 cocaine-exposed and 131 nonexposed) were seen at 1 year (corrected) age 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. All women were identified from a high-risk population screened for drug use. Urine drug toxicology screens were performed by the hospital on women who received no prenatal care, seemed to be intoxicated or taking drugs, had a history of involvement with the Department of Human Services in previous pregnancies, or self-admitted or seemed to be high risk for drug use after interview by a social worker or medical resident. Urine samples were obtained immediately before or after labor and delivery and analyzed for the presence of cocaine metabolites (benzoylecgonine, [BZE]), cannabinoids (THC), opiates, pentachlorophenol (PCP), and amphetamines. The Syva Emit method (Syva Company, 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, ie, BZE, meta-hydroxybenzoylecgonine (m-OH-bze), cocaethylene,[27] cannabinoids (THC), opiates, PCP, amphetamines, and benzodiazepines.[18] Meconium specimens were collected from the newborns' diapers 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 minutes to ensure homogeneity and stored in a refrigerated container. Additional details concerning collection of meconium can be found in a separate report.[19]

Screening assays were conducted using Abbott Diagnostics polarization immunoassay reagents. Cutoff levels for drugs of interest were as follows: cocaine and metabolites, 25 ng/g; opiates, 25 ng/g; amphetamines, 100 ng/g; phencyclidine, 25 ng/g; tetrahydro-cannabinol, 25 ng/g. Confirmatory assays were conducted using gas chromatography-mass spectrometry 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 >the 70th percentile or self-report >the 75th percentile for the users. A previous report[19] on the concordance of meconium concentration and maternal self-report measures of heavier versus lighter use in the entire sample from which the present cohort was formed indicated that significant positive correlations ranging from 0.32 to 0.57 were found between the severity of cocaine use from maternal self-report and the amount of cocaine, cocaethylene, BZE, and m-OH-bze detected in the offspring's' 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.

Procedures

At 1 year old, all infants were administered the PLS-3[28] as part of their follow-up at the child development laboratories. The PLS-3 is a standardized, normative language assessment comprised of Auditory Comprehension and Expressive Communication subscales and consists of receptive and expressive language tasks for ages birth to 4 years 11 months. A summary scale, the Total Language scale, can also be computed. The PLS-3 allows the assessment of language abilities of very young children, including infants, by targeting precursors of later language skills including attention, social communication, and vocalization skills. In addition, it incorporates the use of observations of spontaneous interactions with the child. All examiners were unaware of infant cocaine status.

To assess prenatal drug exposure, infants and their biological mothers were seen as soon as possible after birth, at which time the biological mother was interviewed regarding drug use.[24,29] For the biological mother, for the month before pregnancy and each trimester of pregnancy, mothers were requested to recall frequency and amount of drug use. For tobacco, the number of cigarettes smoked per day was recorded. For marijuana, the number of joints 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 amount of money spent per day were noted. For each drug, the frequency of use was recorded on a Likert-type scale ranging from 0 (not at all) to 7 (daily use), which was then 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 before pregnancy and for each trimester. This score was then averaged for a total score for prenatal exposure for each drug. This assessment was updated at each follow-up visit to provide a similar measure of current drug use, and was also administered to the foster or relative caregiver to provide a measure of postnatal exposure for children placed out of maternal care.

Because introduction of the language measure into the follow-up assessment was initiated midway into the study period, 265 infants (64%) of the total cohort of 415 infants who were still to be seen for the 1-year follow-up were available for language testing. The infants seen at 1 year for the language testing represented 92% of the survivors originally enrolled who remained to be scheduled for follow-up after the introduction of the language measure.

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 birth weight, length, and head circumference. At the enrollment visit, maternal socioeconomic status (Hollingshead) and educational level were calculated. Maternal vocabulary score was measured using the Peabody Picture Vocabulary Test–Revised[30] and 2 subtests of the Wechsler Adult Intelligence Scale–Revised[31] were administered, ie, the Block Design and Picture Completion subtests to obtain an estimate of nonverbal intelligence. The Brief Symptom Inventory[32] is a standardized self-report scale also administered at birth and at the 1-year visit to obtain a measure of severity of psychological distress. It yields a summary score, the Global Severity Index (GSI), which is an indicator of overall stress symptoms. The Hobel Neonatal Risk Index[33] was computed to obtain a measure of neonatal medical complications. Documentation of nonmaternal care was obtained and classified as relative or foster placement. The same measures of vocabulary, nonverbal intelligence, and psychological distress at birth or infant age of 1 year were also obtained from relative or foster caregivers if children had been placed out of maternal care.

All infants and mothers received transportation and a $35 stipend for participation. The institutional review boards of the participating hospitals approved the study, and informed written consent was obtained from caregivers.

Statistical Analysis

All variables with skewed distributions were normalized by log $x + 1$ transformation before analysis, although means and standard deviations are reported in terms of the original distribution. Cocaine-negative and -positive mothers and infants 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 and χ^2 analyses. To assess for possible bias because the language measures were introduced into the research study midway, comparisons were also made on all medical/demographic variables between the portions of the cohort that received the PLS-3 versus those who were not eligible.

Analyses of variance were used to compare the heavier- and lighter-exposed cocaine groups with the nonexposed group to assess group differences in language outcomes, controlling for covariates. Pearson product moment or Spearman rank order correlations were used to assess the relationships of prenatal drug exposure and demographic and medical factors to language outcomes and to determine which control variables would be included in the analyses to control for potential confounding. All control variables (trimester and summary drug measurements for alcohol, marijuana, tobacco, and other drugs, sociodemographic, and maternal IQ variables) which related to the outcome at a level of $P < .10$ and which differed between the exposed and nonexposed groups were statistically controlled.

Both postpartum and concurrent maternal and/or caregiver psychological distress scores and drug use variables were also evaluated as potential confounders of outcome. For infants placed outside maternal care at birth or for the greater part of the first year, foster or kin caregiver education level, socioeconomic status, severity of concurrent drug use, vocabulary, nonverbal intelligence and psychological distress scores were used as covariates in addition to the biological mother's. Multiple and logistic regression analyses were conducted to assess the effects of heavier or lighter cocaine exposure on language outcomes after control for confounders. Potentially mediating variables, specifically Hobel neonatal risk score, Apgar score, birth weight, length, head circumference, and gestational age, were assessed posthoc. Maternal psychological distress was considered separately because it can be both a mediating and a confounding variable of cocaine effects.[34]

RESULTS

Sample Characteristics

Characteristics of the sample are presented in Table 1. Cocaine-using women and controls were primarily black, of low income, and single marital status. The cocaine-using women, both heavier and lighter users, were older, had more children, and received fewer prenatal care visits than nonusers. They used other drugs more frequently and in higher amounts than nonusers. Of users, 66 (49%) were classified as heavier and 68 (51%) as lighter users. Lighter users averaged 6.4 ± 4 (ranging from 0.1–16.5) and heavier users 36.3 ± 40 (ranging from

TABLE 1. Demographic Characteristics

	Cocaine		Noncocaine		
	Heavier $n = 66$	Lighter $n = 68$	$n = 131$	F/χ^2	P Value
	Mean ± SD	Mean ± SD	Mean ± SD		
Biological mother					
Age (y)	29.7 ± 5	30 ± 5	26 ± 5	20.5	.0001‡
Gravida	4.9 ± 2	5.0 ± 3	3.6 ± 2	10.4	.0001‡
Parity	3.5 ± 2	3.4 ± 2	2.7 ± 2	5.4	.005‡
Month began prenatal care	4.8 ± 2	4.2 ± 2	3.2 ± 2	16.4	.0001‡
Number of prenatal visits	5.4 ± 5	5.6 ± 5	9.0 ± 5	15.8	.0001‡
Tobacco use	60 (91%)	49 (80%)	49 (38%)	57.7	.001‡
Alcohol use	54 (81%)	55 (90%)	85 (66%)	8.0	.005‡
Marijuana use	36 (54%)	27 (44%)	18 (14%)	36.5	.001‡
PPVT-R score	71.4 ± 14	75.5 ± 18	77.0 ± 16	4.3	.02§
WAIS-R PC score	6.2 ± 2	6.3 ± 2	7.0 ± 2	1.3	.26
WAIS-R BD score	6.7 ± 2	6.7 ± 2	7.0 ± 2	.42	.67
Race (% nonwhite)	53 (80%)	59 (87%)	106 (81%)	.07	.90
Years of education	11.4 ± 2	11.8 ± 2	12 ± 1	2.9	.06§
Low socioeconomic status	65 (99%)	66 (97%)	129 (99%)	0.02	0.90
GSI score	0.98 ± 0.9	0.65 ± 0.4	0.45 ± 0.5	17.3	.0001†
Current caregiver					
PPVT-R score	71.0 ± 14	75.5 ± 18	78.4 ± 16	4.4	.01§
WAIS-R PC score	6.4 ± 2	6.7 ± 2	6.3 ± 2	1.4	.26
WAIS-R BD score	6.8 ± 2	6.7 ± 2	7.0 ± 2	0.4	.70
Alcohol summary*	2.5 ± 6	3.3 ± 7	1.6 ± 4	2.2	.11
Marijuana summary*	0.3 ± 1	0.6 ± 2	0.8 ± 5	0.5	.62
Concurrent GSI score	0.50 ± 0.7	0.48 ± 0.6	0.37 ± 0.5	1.2	.31

* Average number of cocaine rocks, alcohol drinks or marijuana joints per week during previous 6 months.
† None/heavier and lighter.
‡ None/heavier and lighter.
§ None/heavier.

0.1–175) units (rocks) of cocaine per week over the pregnancy. Both heavier and lighter cocaine-exposed infants were more likely to be preterm and of lower birth weight, head circumference, and birth length than nonexposed infants (see Table 2).

At birth, 49 (37%) exposed infants were placed outside maternal care. These were more likely to be more heavily-exposed infants. In contrast, only 3 (2%) of nonexposed infants were placed. By 1 year, 66 (49%) cocaine-exposed infants were placed outside maternal care, with equivalent distribution of lighter versus heavier exposure, compared with 10 (8%) nonexposed infants. When those infants who were administered the language assessment were compared with those who were not, no differences were found within the cocaine-exposed group on any medical, drug, or sociodemographic factor, except that those given the PLS-3 had more prenatal visits. Within the nonexposed group, there were trends for those given the PLS-3 to have lower maternal Block Design scores. Thus, these differences would have made it less likely to detect differences between the exposed and nonexposed groups.

Relationship of PLS-3 Outcomes to Confounders and Drug Measures

The relationship of outcomes to potentially confounding and mediating variables is shown in Table 3. Boys, both exposed and nonexposed, had lower scores on all domains than girls, but cocaine exposure groups did not differ by gender. Higher maternal parity was related to lower expressive communication scores. Lower biological maternal and concurrent caregiver Block Design scores, and shorter birth length were related to poorer auditory comprehension, but Block Design scores did not differ by group. Other concurrent caregiver measures of vocabulary, intelligence, psychological distress, and severity of drug use were unrelated to any language measure. Placement out of maternal care was unrelated to any language outcome.

Maternal self-report of severity of cocaine use was inversely related to auditory comprehension scores for the month before pregnancy, trimesters 2 and 3, and averaged over pregnancy (See Table 4). There was a nonsignificant trend for trimester 2 marijuana use to relate to better expressive language scores.

The concentration of BZE in infant meconium was inversely related to all outcomes. There were nonsignificant trends for ng/g of m-OH-bze to be negatively related to expressive communication and total language scores.

Language Outcomes

At 1 year of age, more heavily cocaine-exposed infants had poorer auditory comprehension scores than nonexposed infants (see Table 5). Total language scores were also significantly lower, with more heavily exposed infants performing more poorly than both lighter-exposed and nonexposed infants. More heavily exposed infants were also more likely to be classified as mildly delayed in total language scores than nonexposed infants. There were no interaction effects with infant gender, nor were there any mediating effects of infant birth length or maternal psychological distress; ie, the effect of heavier cocaine exposure on outcomes was not reduced when potential mediator or maternal distress scores were included in the regression analysis.

DISCUSSION

The present study found an adverse effect of heavier prenatal cocaine exposure on infant auditory comprehension at 1 year, whereas expressive communication skills were not different from those of lighter or nonexposed infants. More heavily exposed infants were also more likely to be classified as mildly delayed in language skills than nonexposed infants. Findings could not be attributed to other drugs or to a large number of potentially confounding medical, social, and demographic factors, including maternal psychological distress, intelligence, infant placement out of biological maternal care, characteristics of the caregiving environment in nonmaternal care, or postnatal drug exposure. Moreover, poorer auditory comprehension was related to higher concentration of the cocaine metabolite BZE in infant meconium, providing a biological correlate for the group findings. Significant relationships were also found for maternal self-report of higher levels of cocaine use in trimesters 2 and 3 and poorer auditory comprehension scores. As has been generally reported, regardless of exposure status, girls had better language functioning than boys.[35] Prevalence estimates for language disorders in general report a higher incidence for boys (8%) than girls (6%).[36]

TABLE 2. Child Characteristics

	Cocaine		Non-Cocaine		
	Heavier (n = 66)	Lighter (n = 68)	(n = 131)	F/χ^2	p
Birth weight (g)*	2694 ± 625	2714 ± 684	3082 ± 715	12.58	.0001†
Gestational age (wk)	37.8 ± 3	37.6 ± 3	38.3 ± 3	1.4	.23
Birth length (cm)*	47 ± 4	47 ± 4	49 ± 4	7.89	.0005†
Head circumference (cm)*	32.3 ± 2	32.2 ± 2	33.3 ± 2	7.85	.0005†
Apgar—1 min	8.0 ± 2	7.9 ± 2	7.8 ± 2	0.37	.69
Apgar—5 min	8.8 ± 1	8.8 ± 1	8.8 ± 1	0.28	.76
Female	37 (56%)	38 (56%)	69 (53%)	0.24	.87
AGA	54 (83%)	57 (85%)	124 (95%)	15.6	.09†
Hobel score	6.4 ± 14	8.5 ± 18	6.3 ± 16	0.45	.64

* P values adjusted for gestational age.
† None/heavier and lighter.

TABLE 3. Correlations of Maternal and Current Caregiver Demographic and Medical Variables With Outcomes

	Auditory Comprehension	Expressive Communication	Total Communication
Gender	−0.14‡	−0.13‡	−0.16§
Parity	−0.01	−0.16§	−0.10‡
Number of prenatal visits	−0.01	0.10	0.06
Maternal WAIS-R PC score	0.05	−0.02	0.01
Maternal WAIS-R BD score	0.13‡	0.06	0.07
Maternal PPVT-R score	−0.01	0.02	0.01
Maternal education	−0.06	0.03	−0.01
Marital status	−0.06	0.08	0.02
GSI	−0.10†	−0.07	−0.10
Gestational age	0.07	0.03	0.05
Birth weight	0.06	−0.01	−0.02
Birth length	0.12†	0.04	0.09
Head circumference	0.10	0.01	0.06
Apgar 5 minutes	0.07	0.05	0.06
Hobel score	−0.02	−0.05	−0.04
Current caregiver			
PPVT-R score	0.01	0.01	0.01
WAIS-R BD score	0.13‡	0.06	0.11†
WAIS-R PC score	0.05	−0.02	0.01
Cocaine average*	−0.09	−0.05	−0.08
Alcohol average*	0.01	−0.04	−0.03
Marijuana average*	0.05	0.05	0.06
GSI	−0.02	0.05	0.05

* Average number of cocaine rocks, alcohol drinks or marijuana joints per week during previous 6 months.
† $P < .10$.
‡ $P < .05$.
§ $P < .01$.

Cocaine exposure prenatally has been shown to affect neurologic mechanisms that may potentially affect language development, including attention, memory, and motor skills. Alteration of fetal neurotransmitters[37,38] may reduce the speed of processing of auditory information, which has been shown to be associated with language impairment.[39] Cocaine exposure may alter fetal brain development and neuronal formation in the parietal lobe,[40] a brain region associated with auditory word processing.[41] Neurologic alterations may affect arousal and attentional systems,[42,43] as well as memory processes that influence language and cognitive skills. Fetal exposure to cocaine is thought to adversely affect developing regions of the brain regulating arousal and attention.[44] In the present study, for 1-year-old infants the PLS-3 within the Auditory Comprehension subscale evaluates precursors of receptive language skills with tasks that focus on attentional abilities, as the child must attend to objects, people, and language in the environment before language comprehension occurs. Indeed, in other studies of this cohort in the neonatal period, heavier cocaine exposure was associated with poorer visual attention, and recognition memory[15] and more abnormalities in visual and auditory attention.[45] Similarly, in a large longitudinal sample of term, cocaine-exposed, and nonexposed children seen to 5 years old, poorer performance on a latent variable of attentional processing derived from Bayley Mental Scale items through the first 2 years of life was associated with cocaine exposure.[46]

Environmental effects of maternal substance abuse were also considered. Cocaine-using women may have poor parenting skills leading to inability to initiate or sustain affective, social, and linguistic interactions with their children.[47] However, postnatal environmental circumstances that have been demonstrated previously to affect child linguistic outcome were considered in this study, including maternal education, intelligence, vocabulary, and psychological distress symptoms. Both biological maternal and current caregiver nonverbal intelligence were related to 1-year language outcomes, but these effects did not account for differences related to drug exposure. The lack of relationship of most of the measured environmental caregiving factors to infant outcomes at 1 year in this study is consistent with previous work, which has demonstrated that the effects of such factors on child developmental assessments do not usually become apparent until well into the second year of life.[48,49] Moreover, the comparison group was exposed to alcohol, marijuana, and/or tobacco and was drawn from a similarly low socioeconomic status population. Thus, relationships between environmental factors and outcome may have been attenuated in this sample.

Recent research has demonstrated that functional behaviors manifest in infancy such as prelinguistic vocalization, social attention, and communicative gestures form a measurable developmental progression underlying later speech and language.[28,50] Although global assessments of infant cognitive functioning are not predictive of later outcome,[51] there is emerging evidence that specific measures of function may bear greater relationships to later abilities, especially measures of communicative competence.[52]

In contrast to other studies,[53–55] this study did not find a relationship between measures of tobacco ex-

TABLE 4. Correlations of Drug Exposure and One Year Language Outcomes

	Auditory Comprehension	Expressive Communication	Total Language
Meconium			
ng/g Cocaine	−0.04	−0.09	−0.08
ng/g Cocaethylene	0.03	−0.04	0.01
ng/g Benzoylecgonine	−0.15‡	−0.15‡	−0.17§
ng/g M-OH benzoylecgonine	−0.11	−0.12†	−0.12†
Cigarettes per day			
Month prior	−0.02	−0.01	−0.02
Trimester 1	−0.06	−0.07	−0.08
Trimester 2	−0.05	−0.07	−0.07
Trimester 3	−0.03	−0.07	−0.06
Average	−0.04	−0.03	−0.05
Alcohol*			
Month prior	−0.03	0.04	−0.01
Trimester 1	−0.01	0.01	−0.01
Trimester 2	−0.03	0.01	−0.02
Trimester 3	−0.08	−0.03	−0.06
Average	−0.08	0.06	−0.02
Marijuana*			
Month prior	−0.01	0.03	0.02
Trimester 1	0.05	0.08	0.09
Trimester 2	0.03	0.11†	0.10
Trimester 3	−0.02	0.04	0.02
Average	−0.01	0.06	0.04
Cocaine*			
Month prior	−0.12‡	−0.01	−0.08
Trimester 1	−0.09	−0.03	−0.07
Trimester 2	−0.13‡	−0.02	−0.09
Trimester 3	−0.12‡	0.03	−0.04
Average	−0.14‡	−0.01	−0.08

* Average number of cocaine rocks, alcohol drinks or marijuana joints per week during previous 6 months.
† $P < .10$.
‡ $P < .05$.
§ $P < .01$.

TABLE 5. Language Outcomes at One Year

	Cocaine		Noncocaine	F/χ^2	P Value
	Heavier ($n = 66$) M ± SD	Lighter ($n = 68$) M ± SD	($n = 131$) M ± SD		
Chronologic age (wk)	57 ± 6	58 ± 6	57 ± 6	0.2	.30
Corrected age (wk)	55 ± 4	55 ± 6	55 ± 6	0.3	.73
Auditory Comprehension score	86 ± 9	88 ± 10	89 ± 9	3.1	.05*†
Mild delay (%)	22 (33%)	27 (40%)	32 (24%)	2.5	.11
Moderate delay (%)	6 (9%)	6 (9%)	6 (5%)	1.7	.19
Expressive Communication score	91 ± 11	94 ± 9	93 ± 10	1.2	.24‡
Mild delay (%)	27 (41%)	14 (21%)	31 (24%)	3.6	.06‡
Moderate delay (%)	7 (11%)	1 (2%)	5 (4%)	3.3	.07‡
Total Language score	87 ± 10	90 ± 8	90 ± 9	3.1	.02†§‖
Mild delay (%)	25 (38%)	17 (25%)	27 (21%)	6.4	.01†§
Moderate delay (%)	10 (15%)	13 (4%)	9 (7%)	2.9	.09§

Mild = <85 standard score.
Moderate = <77 standard score.
* Adjusted for maternal block design score.
† Heavy/none, $P < .05$.
‡ Adjusted for parity and log trimester 2 marijuana average score.
§ Adjusted for parity and current caregiver block design score.
‖ Heavy/light, $P < .05$.

posure and auditory/verbal comprehension. These differences may be attributable to the difference in methodology between studies, as findings for tobacco exposure were found using clusters of items from the Bayley Scales[55] and at older ages.[53,54]

The present study also demonstrated differences between the relationship of maternal self-report measures of cocaine use and the concentration of cocaine metabolites in meconium to expressive communication and total language scores that may reflect differences in timing of exposure or reliability of self-report. The use of both self-report and meconium measures to categorize exposed infants into heavier and lighter categories may have enhanced reliability in this study and allowed detection of significant differences.

The present study demonstrates significant behavioral teratogenic effects of heavier cocaine exposure on auditory comprehension skills underlying speech-language development in the first year of life. Early identification of such delays can lead to intervention services demonstrated to help in facilitating appropriate language development.[56] Pediatricians are in a unique position to monitor infant development during ongoing well-child care visits in the first 2 years of life. The Committee on Practice and Ambulatory Medicine of the American Academy of Pediatrics recommends that pediatricians provide developmental monitoring through the process of surveillance in which performing skilled, longitudinal observations of children is emphasized.[54] Although in the present study a standardized assessment of skills was used, most of the items on which more heavily exposed infants were delayed can be observed during a pediatric visit, or can be evaluated via parental report. These include localizing to sounds, visually following an object, attending to toys or books, and playing social games.[57] Because even the relatively small effects on attention and auditory comprehension of heavier cocaine exposure documented in this study can have large population effects on the numbers of children needing long-term intervention services,[58] increased developmental surveillance of cocaine-exposed infants by pediatricians is needed.

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INDEPENDENT EVALUATION OF "EXPERT" TESTIMONY

Pathological science results from scientists fooling themselves. Junk science is more sinister; it is deliberately designed to fool or befuddle nonscientists, particularly juries . . . In 1993, the Supreme Court ruled that [the latter] testimony is not credible and instructed federal judges to serve as "gatekeepers," aggressively screening out ill-founded or speculative theories . . . For example, an Oregon federal district court appointed a special panel of 4 independent scientists to evaluate expert testimony in some 70 cases involving silicone breast implants.

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