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ARTICLE

Prenatal Cocaine and Tobacco Effects on Children's Language Trajectories

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ABSTRACT

OBJECTIVE. The objective of this study was to examine the effects of prenatal cocaine and polydrug exposure on language development of preschool children using a prospective longitudinal model, controlling for confounders.

METHODS. Children who were exposed to cocaine in utero ($n = 209$) and nonexposed children ($n = 189$) were followed prospectively at birth and at 1, 2, 4, and 6 years of age and were compared on receptive, expressive, and total language scores across time using random coefficient models, controlling for confounders.

RESULTS. A significant, stable effect of cocaine exposure on language development was observed over time for all language domains, with cocaine exposure related to poorer language performance. Cigarette exposure was related to lower receptive language scores. Environmental influences on language scores were also observed. Both the cocaine-exposed and nonexposed children declined in language performance over time.

CONCLUSIONS. Prenatal cocaine exposure has a stable negative effect on language skills during the first 6 years of life. Both cocaine-exposed and nonexposed children showed decreased language growth over time; however, cocaine-exposed children demonstrated linguistic deficits compared with nonexposed peers and did not catch up. Cigarette and environmental influences were also noted.

CHILDREN WHO ARE exposed to cocaine in utero are at risk for a variety of developmental delays as a result of both biological risk and postnatal environmental influences. Biological risks include a disruption in arousal and attention.[1–5] The environmental risks are threefold: inadequate stimulation provided by a drug-using mother,[6,7] insecure child attachment,[8] and poverty.[9] These risk factors may have an impact on the language development of children who are exposed to cocaine.

Outcome studies of language development of children who were exposed to cocaine in utero have been equivocal,[4,10–20] possibly as a result of methodologic differences such as age at assessment, instruments used, and variability in selection of confounding factors considered.[15] Although most studies have reported language outcomes at a single time point, one of the few longitudinal prospective studies of children who were exposed to cocaine found stable cocaine-specific effects on total language scores, even after control for multiple medical and demographic covariates.[11,21] Children with cocaine exposure performed on average 15% of a SD lower on a standardized language test than nonexposed children, with the strongest effects at 18 months and 3 years. This study extended these findings with a larger developmental age span, examining also receptive and expressive language and using biological markers (meconium) of cocaine exposure.

This investigation examined the effects of cocaine exposure in utero on language development in a longitudinal sample of children who were enrolled prospectively at birth and followed to 1, 2, 4, and 6 years of age. Children who were exposed to cocaine in utero were hypothesized to perform more poorly than nonexposed children on standardized measures of receptive and expressive language across all time points. Careful delineation of environmental factors that are known to relate to child language skills was conducted, particularly maternal education/vocabulary, depressive symptoms, and use of other substances.

METHODS

Subjects

A total of 398 children (209 cocaine exposed [CE] and 189 non–cocaine exposed [NCE]) were followed prospectively from birth and assessed for language development at 1, 2, 4, and 6 years of age. The sample was drawn from a cohort that was recruited at birth from a large, urban, county teaching hospital to participate in a longitudinal study of the sequelae of fetal drug exposure. Approval from University Hospitals of Cleveland Institutional Review Board for Human Investigation and informed consent were obtained. Women who were considered at high risk for drug use were administered drug toxicology screenings. Urine samples were obtained immediately before or after labor and delivery and analyzed for the presence of cocaine metabolites (benzoylecgonine), cannabinoids, opiates, phencyclidine, and amphetamines. In addition, infants had meconium drug analyses performed for cocaine and its metabolites (benzoylecgonine [BZE], meta-hydroxybenzoylecgonine [M-OH-BZE], cocaethylene, cannabinoids, opiates, phencyclidine, amphetamines, and benzodiazepines). CE infants were identified on the basis of positive infant meconium, maternal urine, or maternal self-report, whereas control infants were negative on all indicators. Women who used alcohol, marijuana, or tobacco during pregnancy were included in both groups.

The sample size for the original cohort was 415. The number of children who participated in the language testing at each follow-up time point varied; however, ~85% of the cohort participated in 3 or more of the 4 visits. At 1 year, the sample size consisted of 405 children, with 371 assessed and 265 children receiving the language measures. At 2 years, 404 children from the original sample were available (1 death), with 381 assessed and 339 completing language measures. At 4 years, 404 children were available, with 394 completing the language test, 12 (8 CE) not coming to the visit, 16 (12 CE) dropouts, and 2 (1 CE) moving out of state. Attrition was greater for the CE group ($P = .04$) than for the NCE group. At 6 years of age, 377 received assessments, with 371 children completing the language test battery.

Procedures

At 1 and 2 years of age, the Preschool Language Scale, Third Edition[22] was administered. At 4 years, the Clinical Evaluation of Language Fundamentals–Preschool[23] was given. At 6 years, the Comprehensive Assessment of Spoken Language[24] was administered. At all ages, examiners were unaware of infant cocaine status.

For assessment of prenatal drug exposure, infants and their biological mothers were seen immediately after birth, at which time the biological mother was interviewed regarding drug use. Biological mothers were asked to recall the frequency and the amount of drug use for the month before pregnancy and each trimester of her pregnancy. More specific, for tobacco, the number of cigarettes smoked per day was recorded; for marijuana, the number of joints smoked per day was recorded; for alcohol, the number of drinks of beer, wine, or hard liquor per day was computed; and for cocaine, the number of rocks consumed and amount of money spent per day were noted. 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 environmental exposure for children who were 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, and type of medical insurance, infant Apgar scores, birth weight, length, and head circumference. At enrollment, maternal socioeconomic status (SES; A. B. Hollingshead, PhD, *Four-Factor Index of Social Status*, unpublished manual, 1975) and educational level were calculated. Maternal vocabulary score was measured using the Peabody Picture Vocabulary Test, Revised (PPVT-R).[25] Two subtests of the Wechsler Adult Intelligence Scale, Revised[26] (WAIS-R) were administered: The Block Design and Picture Completion subtests from the WAIS-R enabled an estimation of nonverbal intelligence. The Brief Symptom Inventory[27] is a standardized self-report scale that was administered at birth and at all visits to obtain a measure of severity of psychological distress. The General Severity Index, a summary score of the Brief Symptom Inventory, was used as an indicator of overall distress. The Hobel Neonatal Risk Index[28] was computed to obtain a measure of neonatal medical complications. Also at the visit, the child's placement (either biological mother/relative or foster/adoptive caregiver) was noted, and data on the current caregiver were updated. When the child had been placed with a new caregiver, intellectual measures of the caregiver 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.[29]

Statistical Analysis

Baseline maternal and child characteristics and prenatal drug exposure were summarized using means and SDs for continuous variables and frequencies and percentages for categorical variables. Comparisons between CE and NCE groups were performed using *t* tests, Wilcoxon rank sum tests, and Pearson χ^2 tests. All positively skewed data, including drug self-report measures and General Severity Index, were transformed using the natural logarithm of $(x + 1)$ to achieve a distribution that approximates normality. Correlations between drug exposure data and language outcomes were estimated using Spearman correlation coefficients.

For examination of language performance across time, each measurement was internally standardized to create *z* scores at each visit using all available children. Standardization of scores at each time point allowed modeling to be performed across language measures despite that different age-appropriate tests were used. Analyses of the *z* scores were accomplished using random coefficient models with restricted maximum likelihood estimation. The intercept and the slope for child age were treated as random effects to capture the variability and correlation in the data. The actual age of the child was used instead of visit age to capture trends over time better. These models were used to estimate and test relationships between cocaine exposure groups at the follow-up visits (ages 1, 2, 4, and 6 years). Initially, child age, cocaine exposure, and the interaction between age and exposure were included in the model to test for changing effects over time. Because the interaction term was not significant for any of the language measures, main effects models were fit. The developmental trajectories were allowed to be nonlinear (eg, quadratic) using polynomials of time. The lowest order polynomial of time was retained. In all models, the trajectories were found to be linear. The effects of cocaine are presented with and without consideration of possible confounding variables.

For each measure, the model-building strategy of Bandstra et al[11] was followed to achieve a final model that controlled for possible confounding and moderating variables to estimate an unbiased effect of prenatal cocaine exposure on language development. The following variables were considered for each outcome: child's age, prenatal cocaine exposure (yes versus no), and the interaction between age and cocaine exposure; maternal age at child's birth, current caregiver's PPVT-R and WAIS-R block design; child's race and child's gender; prenatal drug variables (alcohol, cigarettes, and marijuana), prenatal care, parity, SES, and marital status; current HOME scale; and adoptive/foster care. Adjusted least squares means and SEs were calculated from the final models and compared between treatment groups at each follow-up visit age. Plots of the adjusted group means ($\pm$SE) for each language measure over time are provided. Analyses were performed using SAS 9 (SAS Institute, Cary, NC).

RESULTS

Sample Characteristics

Cocaine-using women and control subjects were primarily black, of low income, and not married (Table 1). Cocaine-using women were older, had more children, and attended fewer prenatal care visits. They used other drugs more frequently and in higher amounts than nonusers (Table 2). CE infants were more likely to be preterm and of lower birth weight, head circumference, and birth length than NCE infants (Table 3).

At birth, 49 (26%) CE infants were placed outside maternal/biological care compared with only 3 (2%) of NCE infants. By 4 years, 42 (22%) CE children were in adoptive/foster care compared with 10 (8%) of NCE children. CE children averaged 1.0 $\pm$ 0.99 nonmaternal care placements by 6 years versus 0.16 $\pm$ 0.48 for NCE infants ($t = -10.12$; $P < .0001$). There were no group differences in HOME environment scores.

From birth to 6 years, there were 11 deaths (8 CE and 3 NCE; $\chi^2 = 1.9$, $P < .17$). Causes of death for the CE children were sudden infant death syndrome ($n = 4$), cardiopulmonary arrest ($n = 1$), pneumonia ($n = 1$), accidental asphyxia ($n = 1$), and respiratory distress syndrome ($n = 1$). Causes of death for the NCE children

TABLE 1 Maternal Demographics for CE and NCE Groups

Maternal Demographics	CE Group (N = 209)		NCE Group (N = 189)		P
	Mean	SD	Mean	SD	
Age at birth	29.68	5.00	25.71	4.90	<.0001
Years of education	11.57	1.65	11.99	1.41	.007
Parity	3.53	1.87	2.71	1.85	<.0001
No. of prenatal visits	5.23	4.62	8.78	4.87	<.0001
PPVT-R	73.78	15.20	77.82	14.86	.009
WAIS-R Design Scale	6.86	2.18	7.15	2.12	.20
WAIS-R Picture Completion	6.60	2.14	7.02	2.38	.07
Global Severity Index	0.82	0.73	0.49	0.53	<.0001
Black, n (%)	169	(82.04)	153	(80.95)	.78
Currently employed, n (%)	11	(5.37)	40	(21.28)	<.0001
Married, n (%)	16	(7.77)	32	(16.93)	.005
Low SES, n (%)	201	(98.05)	185	(97.88)	.91

TABLE 2 Maternal Drug Use During Pregnancy for CE and NCE Groups

Maternal Drug Use During Pregnancy	CE Group (N = 209)		NCE Group (N = 189)		P
	Mean	SD	Mean	SD	
Tobacco, cigarettes per d	11.57	10.95	4.11	7.62	<.0001
Alcohol, dosage per wk	10.16	18.71	1.35	4.52	<.0001
Marijuana, dosage per wk	1.36	3.60	0.59	3.45	<.0001
Cocaine, units per wk	23.56	44.62			
Prevalence of Use, n (%)					
Tobacco	174	(87.88)	74	(40.88)	<.0001
Alcohol	171	(86.36)	119	(65.75)	<.0001
Marijuana	96	(25.33)	23	(12.71)	<.0001
Amphetamine	5	(2.54)	2	(1.11)	.31
Barbiturate	1	(0.51)	1	(0.55)	.95
Heroin	5	(2.55)	0	(0)	.03
Phencyclidine	11	(5.64)	0	(0)	.001

TABLE 3 Child Demographics for CE and NCE Groups

Child Demographics	CE Group (N = 206)		NCE Group (N = 189)		P
	Mean	SD	Mean	SD	
Birth weight, g[a]	2726	642	3100	691	<.001
Birth length, cm[a]	47.36	3.89	49.10	3.68	<.0001
Head circumference, cm[a]	32.31	2.13	33.46	2.35	<.0001
Microcephaly	30	14.78	10	5.35	.002
Apgar score (1 min)	8.00	1.41	7.93	1.67	.61
Apgar score (5 min)	8.79	0.64	8.78	0.70	.89
Hobel Neonatal Risk score	7.27	16.28	5.67	15.57	.32
Male, n (%)	94	(45.63)	92	(48.68)	.54
Black, n (%)	168	(81.55)	152	(80.42)	.77
Preterm (<37 wk gestational age), n (%)	58	(28.16)	35	(18.52)	.02
Low birth weight (<2500 g), n (%)	72	(34.95)	34	(17.99)	.0001
Very low birth weight (<1500 g), n (%)	12	(5.83)	7	(3.70)	.33
Small for gestational age, n (%)	25	(12.32)	4	(2.13)	.0001

[a] Adjustment for gestational age to these measures.

were sudden infant death syndrome (n = 2) and respiratory distress syndrome (n = 1). From enrollment at birth, the retention rate was 93% (377) at 6 years for surviving children.

Meconium Assays and Outcomes

Several significant relationships were found between the concentration (ng/g) of cocaine metabolites and child language outcomes. At 1 year, the concentrations of BZE ($r = -0.16$, $P < .02$) and M-OH-BZE ($r = -0.14$, $P < .04$) were negatively related to the expressive language score; and at 2 years, the concentration of BZE was negatively related to receptive ($r = -0.14$, $P < .02$), expressive ($r = -0.12$, $P < .05$), and total language scores ($r = -0.11$, $P < .02$). At 4 years, the concentration of cocaethylene, the metabolite formed through the

combination of cocaine and alcohol, was negatively related to expressive language ($r = -0.12$, $P < .03$) and marginally related to total language score ($r = -0.11$, $P < .06$). By 6 years of age, there were no significant relationships.

Longitudinal Analyses

Table 4 presents the mean unadjusted receptive, expressive, and total language scores by group for each of the 4 time points. Table 5 presents the adjusted mean language scores at each visit for the CE and NCE groups.

Total Language

The interaction between child age and cocaine exposure was not significant, suggesting that total language development demonstrated a similar growth trajectory across the groups. In the final model, a main effect for race was found, indicating that black children scored almost 0.5 of a SD lower than nonblack children. Significant interactions of child age with maternal vocabulary score, maternal age, and child gender were found. Gender was associated with total language scores at 1, 2, and 4 years of age, with boys performing more poorly. Maternal vocabulary was significant at 4 and 6 years, with higher maternal vocabulary related to higher child total language scores. Older maternal age at the child's birth was significant only at 6 years, with older maternal age related to poorer language scores. Controlling for these covariates, the effect of cocaine exposure was significant ($P = .0385$), such that CE children had a mean standardized total language score of 0.18 SD (SE = 0.09) lower than NCE children at all time points (Fig 1). Alcohol and marijuana effects were not significant; however, cigarette exposure was related to lower total language scores. Also, prenatal care, SES, and marital status were not related to total language scores at any of the individual time points or the overall trajectory.

Receptive Language

Similar to total language scores, the interaction between child age and cocaine exposure was not significant for receptive language. Main effects for race and gender were found, such that black children scored more poorly than nonblack children and boys more poorly than girls. Growth in receptive language varied by current HOME score. Higher HOME scores were associated with better receptive language at 4 years of age ($P < .0001$). Controlling for these covariates, a borderline significant effect of cocaine exposure was found ($P = .058$). CE children performed ~0.15 SD (± 0.08) lower than NCE children. No effects of marijuana or alcohol exposure were found. However, cigarette exposure was negatively associated with receptive language ($P = .0168$). Marital status and adoptive/foster care were not significantly related to receptive language.

Expressive Language

The interaction between child age and cocaine exposure was not significant for expressive language scores. Significant main effects for race and gender were found. Black children scored approximately one third of a SD lower than nonblack children, and boys performed one third of a SD lower than girls. The expressive language trajectory also varied by current HOME score ($P = .0002$). Higher HOME scores were associated with higher expressive language scores at 2 and 4 years of age ($P < .0001$). Controlling for these covariates, a significant and constant effect of cocaine exposure was found ($P = .048$). CE children perform ~0.15 SD (± 0.08) lower than NCE children. Marijuana, alcohol, or cigarette exposure was not a significant confounder. Marital status and adoptive/foster care were not related to expressive language development. On average, all children declined in their expressive language scores at 4 years compared with the 1-year visit ($P = .03$).

Associations of the PPVT-R, HOME, and Cigarette Exposure to Language

The effect of current caregivers' PPVT-R was significant at 4 years of age, such that as the PPVT-R scores increased, so did the child's scores on receptive ($P = .0066$) and total language ($P = .0025$) measures. This effect was also observed for the total language score at 6 years of age ($P < .001$).

The HOME scale showed significant effects across the ages. At 1 year of age, the receptive language score was negatively related to the HOME scale, such that as the HOME score increased, the receptive language score decreased ($P = .04$). At 2 years of age, the HOME score was positively related to expressive language, such that as the HOME score increased, so did the child's expressive language score ($P = .037$). At 4 years of age, the HOME score was positively related to all language scores (receptive, $P < .0001$; expressive, $P < .0001$; and total, $P = .005$ scores). This effect remained at 6 years of age ($P = .001$).

Children who were exposed to any cigarette smoking

TABLE 4 Unadjusted Language Scores at Each Time Point for CE and NCE Groups

Language Outcome Scores, Mean (SD)	Age, y			
	1 ($n = 265$)	2 ($n = 340$)	4 ($n = 374$)	6 ($n = 371$)
Receptive				
CE	87 (9)	85 (11)	77 (15)	NA
NCE	89 (9)	88 (13)	80 (17)	NA
Expressive				
CE	92 (10)	87 (12)	83 (12)	NA
NCE	93 (10)	90 (13)	85 (14)	NA
Total				
CE	89 (9)	84 (12)	79 (13)	88 (12)
NCE	90 (9)	88 (12)	82 (14)	90 (15)

NA indicates not applicable.

TABLE 5 Adjusted Mean Standardized Language Scores at Each Visit for CE and NCE Groups

Parameter	CE Group		NCE Group	
	Mean ± SE	95% CI	Mean ± SE	95% CI
Total language scores[a]				
1 y	−0.09 ± 0.07	−0.23 to 0.05	0.09 ± 0.06	−0.04 to 0.22
2 y	−0.10 ± 0.06	−0.22 to 0.03	0.08 ± 0.06	−0.04 to 0.20
4 y	−0.12 ± 0.05	−023 to 0.004	0.06 ± 0.06	−0.06 to 0.18
6 y	−0.14 ± 0.06	−0.27 to 0.01	0.04 ± 0.07	−0.10 to 0.18
Receptive language scores[b]				
1 y	−0.06 ± 0.06	−0.19 to 0.06	0.08 ± 0.07	−0.05 to 0.22
2 y	−0.10 ± 0.05	−0.21 to 0.004	0.04 ± 0.06	−0.07 to 0.16
4 y	−0.18 ± 0.07	−0.31 to −0.05	−0.04 ± 0.07	−0.17 to 0.10
Expressive language scores[c]				
1 y	−0.03 ± 0.07	−0.16 to 0.10	0.12 ± 0.07	−0.01 to 0.26
2 y	−0.09 ± 0.06	−0.20 to 0.03	0.07 ± 0.06	−0.05 to 0.18
4 y	−0.19 ± 0.07	−0.33 to −0.06	−0.04 ± 0.07	−0.17 to 0.09

CI indicates confidence interval.

[a] Adjusted for child age, maternal age, maternal age × child age, current PPVT-R, current PPVT-R × child age, child race, gender, gender × child age, tobacco exposure, current HOME score, and current HOME × child age.

[b] Adjusted for child age, child race, gender, tobacco exposure, current HOME score, and current HOME × child age.

[c] Adjusted for child age, child race, gender, parity, current HOME score, and current HOME × child age.
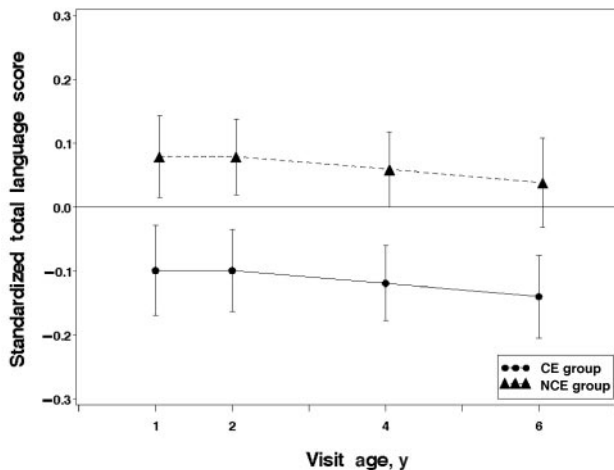

FIGURE 1
Standardized total language score at each visit for CE and NCE groups.

in utero had a lower mean standardized receptive language score by 0.21 ± 0.09 SD (95% confidence interval: −0.38 to −0.04). Children who were exposed to cigarette smoking also had a lower mean standardized total language score by 0.17 ± 0.09 SD (95% confidence interval: −0.35 to 0.01). Exposure to cigarette smoking did not have a significant effect on expressive language at any time point and was therefore not included in the final model for expressive language.

DISCUSSION

This study presents a stable picture of the negative effects of prenatal cocaine exposure on language skills during the first 6 years of life. Correlations of the concentration of cocaine metabolites in infant meconium with later language outcomes also indicated that cocaine exposure affects language development. CE children had poorer language scores at all 4 ages than the NCE children. Although the CE group showed stable language growth over time, they did not catch up to their NCE peers. These findings are consistent with and extend the findings of Bandstra et al,[11] who also found a stable cocaine-specific effect on total language scores between the ages of 3 and 7 years. We examined receptive and expressive language scores at 3 time points and found marginally significant effects of cocaine exposure over time. These findings concur with previous reports of both receptive[5,12,14,30,31] and expressive language delays in children who were exposed to cocaine.[10,13,14]

Of note is that language scores in both the CE and NCE groups declined over time, suggesting that factors that are common to both groups, such as low SES, education, and poverty, have a negative impact on the developmental trajectory of language, a finding that is in agreement with the longitudinal studies of the Miami Prenatal Cocaine study[21] that also report a decline in scores on standard language measures with time. Cigarette smoking during pregnancy also had a negative impact on receptive language skills.

These findings are consistent with previous research by Fried and Watkinson[32] that showed reduced auditory processing skills in children who were exposed prenatally to tobacco. In the neonatal period, those infants demonstrated decreased rates of auditory habituation.[33] At 12 to 24 months, infants showed poorer responses to auditory-related items on the Bayley Scales of Infant Development.[34] Follow-up at 3 and 4 years of age revealed deficits in language skills and at 6 years in auditory processing skills.[32,35] At 9 to 12 years, these children presented with lower language and reading scores, particularly related to the auditory aspects of these skills.[36] The present findings indicate

that tobacco exposure is additive to the risk of CE children for language deficits.

Environmental Effects

Our data also support the notion that environmental variables can affect language skills to a considerable extent. That current caregiver's vocabulary score and the HOME score both seem to have an impact on language performance underscores the environmental modifiability of language. Bandstra et al[11] also found a relationship between language outcomes and the HOME scale. Previous reports of delay in semantic representation in children who were exposed to cocaine[37] may relate to the caregiver's vocabulary rather than to the cocaine exposure itself. The current caregiver's vocabulary score was significant only at the 4-year and 6-year testing times but not at the younger ages. At a young age, the child's language development may not be as influenced by the caregiver's vocabulary because the child is acquiring basic vocabulary and syntactic structures. However, as language development proceeds, the child is required to master complex syntax, and vocabulary growth is extremely rapid. That a caregiver's verbal skills played an important role in the child's language growth is interesting. As reported previously,[3] the foster/adoptive caregiver's vocabulary, depression, and HOME scores mediate the adoptive-care effect on language at 4 years of age.

Limitations of Study

Several limitations of this study should be noted. First, not all children were assessed at each time point, with the fewest number of children completing the language test at 1 year of age. However, 85% of the enrolled sample are represented in at least 3 time points. A second limitation was the use of different language measures at different time points. The preschool years are a time of rapid language acquisition, and few measures can adequately assess language skills from 1 year to 6 years of age. Ceiling and floor effects are problematic. Despite that different assessments were used at each time point, the magnitude of language deficit between the CE and NCE children remained constant at each time. This finding suggests that the different measures were assessing similar language constructs.

CONCLUSIONS

The findings from this study support the notion that language outcomes are the result of both drug exposures and environmental factors. The cumulative risk for language disorders is likely to be based on prenatal drug or other toxic exposure, environmental and genetic influences, and social factors. Future studies should attempt to identify specific linguistic deficits in semantic, syntactic, phonologic, and pragmatic skills that are associated with cocaine and tobacco exposures and determine whether specific deficits change over time. Pediatricians should be aware of the additional risks that prenatal cocaine and tobacco exposures have on language development of poor, urban children and increase their surveillance and intervention efforts in these populations.

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