Children Prenatally Exposed to Cocaine: Developmental Outcomes and Environmental Risks at Seven Years of Age

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ABSTRACT. Data are equivocal regarding the long-term consequences of prenatal exposure to cocaine on school-aged children. We compared 101 children exposed prenatally to cocaine with 130 unexposed children on measures of intelligence, visual motor, and motor abilities at age 7 years. Bivariate analyses revealed that cocaine-exposed children scored significantly lower than comparison children on the abbreviated Wechsler Intelligence Scale for Children-Third Edition Verbal and Full Scale IQ scores, the Visual Motor Integration and Motor Coordination standardized scores, and the Bruininks-Oseretsky Fine Motor Composite score. Regression analyses indicated that the biological mother's vocabulary and home environment assessed at the same 7-year visit were stronger predictors of developmental outcome than prenatal drug exposure. Level of cocaine exposure, however, predicted visual motor and motor skills. The results indicate that although prenatal cocaine exposure may confer some degree of developmental disadvantage in the visual motor domain, it frequently occurs in the context of an inadequate rearing environment, which may be a stronger determinant than prenatal cocaine exposure of children's outcome. *J Dev Behav Pediatr 25:83–90, 2004.* Index terms: *cocaine, development, children, caregiving environment.*

Data from a recent national household survey on drug abuse indicate that cocaine use has subsided from the epidemic levels of the late 1980s and early 1990s.[1] The same data, however, indicate that cocaine use, particularly among women, continues to exist and presents as an ongoing, if somewhat diminished, problem.

Through both direct (i.e., drug transfer across the placental barrier) and indirect mechanisms, including uterine artery vasoconstriction and maternal cardiovascular and neurological effects,[2] prenatal exposure to cocaine has the potential to produce both specific and broad effects. A recent review of research on neonatal behavioral and cranial ultrasonography findings[3] concluded that there are measurable but subtle effects of in utero cocaine exposure on infant central nervous system structure and function. Whether these early effects presage neurobehavioral deficits in later child development has yet to be determined.[4]

In some of the earliest follow-up studies, investigators[5–7] argued that prenatal exposure to cocaine has an indirect effect on cognitive ability and a direct effect on behavioral development. From a study of 95 cocaine/polydrug-using

mothers and their children matched to 75 unexposed children, Chasnoff et al[6] reported that mean IQ scores, although at the lower end of the normal range, did not differ between groups of children aged 4 to 6 years. The authors concluded this was an indirect effect of drug exposure mediated by the home environment, particularly concurrent parental drug use. In contrast, children prenatally exposed to drugs displayed clinically significant levels of behavior problems, as documented on the Child Behavior Checklist, that were not directly related to the quality of the home environment. Although Chasnoff et al interpreted this finding as evidence of a direct effect of prenatal exposure to cocaine and other drugs, these findings were presented as a brief review that were not peer reviewed and therefore should be interpreted with caution.

A few studies have examined specific areas of development in cocaine-exposed infants and children. In a separate cohort in which cocaine exposure was determined through both self-report and biological means (meconium and urine), Singer et al[8] found significant behavioral teratogenic effects of fetal cocaine exposure on a precursor of receptive language after controlling for numerous confounding variables. This study found that more heavily exposed infants had lower auditory comprehension scores than non-exposed infants and lower language scores than lighter exposed and non-exposed infants. In Singer et al's cohort at 24 months of age, cocaine exposure accounted for a significant difference in cognitive scores on the Bayley Scales of Infant Development.[9] The 5.8-point difference in Mental Development Index scores and the doubling of incidence of significant cognitive delay indicate the likelihood of later cognitive deficits.

Another study reported that early hypertonia identified in cocaine-exposed children resolved as they matured.[10] Similarly, Kilbride et al[11] found increased muscle tone in cocaine-exposed infants at birth, which apparently resolved by age 12 months with no difference in mean scores between exposed and unexposed children at age 36 months in motor development. In contrast, Arendt et al[12] reported that different drug exposures accounted for significant variance in fine and gross motor development of children at 2 years of age, in that prenatal cocaine exposure predicted poorer fine motor development skills, particularly hand use and eye-hand coordination, whereas prenatal alcohol exposure had an effect on gross motor receipt and propulsion skills.

In a unique approach, Koren et al[13] followed a cohort of 23 children exposed in utero to cocaine who were adopted by middle-to-upper class families. To form a comparison group, adoptive mothers were paired with women attending the same clinic. After socioeconomic class, IQ, and age of the child were controlled for, the adopted group showed a direct neurotoxic effect of cocaine on language and a trend toward decreased IQ at 34 months of age. The effects of drug exposure were more pronounced in cocaine-exposed children who were raised by their biological mothers.

In contrast, studies of Hurt and colleagues[14,15] reached different conclusions regarding drug exposure in 30-month-old children. In this study, children who were exposed in utero to cocaine and other drugs were compared with a control group recruited from the same medical center.

Although Mental Development and Psychomotor Development Index scores decreased over time and were lower than mean scores published for the scales, no drug effect was obtained. The authors concluded that risk of poorer developmental outcome in this cohort was related to socioeconomic and minority status rather than drug exposure, suggesting an environmental rather than biological effect. From data collected on this same high-risk cohort at ages 3.5 and 4.5 years, the authors further concluded that both the cocaine-exposed and control children may experience problems functioning in more cognitively demanding situations, such as a classroom.[16]

In a well-controlled, longitudinal study, Richardson[17] reported on both cognitive and neuropsychological development in a large cohort of predominantly African-American subjects. When the subjects were aged 3 years, Richardson reported that first trimester prenatal cocaine exposure was associated with reduced head circumference and lower composite IQ score on the Stanford-Binet Intelligence Scale. A second study involving a slightly different cohort of 523 non-exposed and only 28 cocaine-exposed children found no significant effects of prenatal cocaine exposure on growth, intellectual ability, academic achievement, or teacher-rated classroom behavior.[18] Children prenatally exposed to cocaine did, however, show deficits in attention on a computerized vigilance task. Thus, equivocal effects of cocaine have been found for physical growth and development.

Although these previous studies are important initial attempts to examine drug effects, they are somewhat limited methodologically. Chasnoff et al's[6] exposed group may represent a unique sample, because the participants were identified during pregnancy and offered drug treatment. Richardson's[17] follow-up sample included only a small number of cocaine-exposed children. Hurt and colleagues'[14,15] findings of an environmental effect may have been attributable to the use of broad measures, a restricted range of assessed developmental domains that were not sensitive to a drug effect, and substantial attrition effects.

To advance beyond these limitations, the present study was designed to investigate the effects of prenatal cocaine exposure on long-term cognitive, visual motor integration, and motor development in a large cohort of 7-year-old children. The primary hypothesis was that 7-year-old children who were prenatally exposed to cocaine would perform less well than a demographically comparable group of unexposed children on standardized measures. In addition, cocaine exposure would account for a significant proportion of the variance in outcomes independent of confounding factors, such as sociodemographic characteristics and exposure to other drugs.

METHODS

Participants

This study was designed to take advantage of an existent clinic in a large urban pediatric hospital. Participants were recruited from either a newborn nursery at delivery in the same urban hospital or at a well-baby visit to this high-risk clinic. The high-risk clinic was established in 1990 by the hospital to address the large increase in deliveries involving

prenatal cocaine exposure. This clinic also served new mothers who were at risk of drug involvement, primarily because they obtained three or less prenatal visits. In general, infants were seen in this clinic by nurse practitioners and were regarded as healthy. Newborns with very low birth weight (birth weight <1500 g), congenital abnormalities, or serious medical conditions were excluded, as were families that did not speak English as a primary language. Social workers and nurses having contact with the families in the hospital and the high-risk follow-up clinic alerted the principle investigator to the fact that the cocaine-exposed patient met inclusion criteria. After the patient's chart was reviewed, all cocaine-exposed families were approached regarding participation in this study during their hospital stay (birth cohort) or during a well-baby visit to the high-risk clinic (cohort of 1 and 2 year olds). Unexposed children were recruited at the same time using the same exclusion criteria. A total sample of 231 (101 cocaine-exposed, 130 unexposed) children from the original sample of 267 who attended the initial study entry visit were seen for testing at the 7-year time point, representing a retention rate of 86.5%. The children seen at the 7-year data collection point, therefore, ranged in age at recruitment from birth (n = 119) to 1 year (n = 65) to 2 years (n = 47) at study entry.

In an effort to examine the potential bias that was present in our recruitment strategy, differences between children recruited at birth, 1 year, and 2 years were examined for all outcome variables. At age 7 years, there were no differences in Verbal IQ (birth = 87, 1 year = 86, 2 years = 85), Performance IQ (birth = 86, 1 year = 83, 2 years = 82), Full Scale IQ (birth = 86, 1 year = 83, 2 years = 82), or any of the other outcome measures. In addition, the recruitment cohort was entered into regression analyses for all outcome variables but was not retained because of lack of significance. Therefore we would argue that these samples could be legitimately combined in subsequent analyses.

Inclusion criteria for this study were as follows: All infants had birth weights of at least 1500 g at delivery and no known genetic abnormalities or HIV, and mothers were at least 16 years of age at time of delivery and did not use either prescribed or street drugs other than alcohol, cigarettes, marijuana, or cocaine. Drug exposure was determined by a combination of medical chart review, maternal or infant urine toxicology results, and clinical interviews. No notation was made regarding the number of mothers who had positive toxicology screens and those who simply reported cocaine use in the birth cohort, with all 1- and 2-year-old children classified as cocaine-exposed on the basis of maternal report or urine screen ordered by the physician attending the birth. All non-exposed children were selected from the same urban hospital high-risk clinic.

MATERIALS

Child participants were assessed on the Developmental Test of Visual Motor Integration (VMI) Fourth Edition,[19] the Bruininks-Oseretsky Test of Motor Proficiency (BOTMP),[20] and an abbreviated form of the Wechsler Intelligence Scale for Children-Third Edition (WISC-III).[21] Examiners trained to criteria and blinded to drug exposure status administered all measures.

The VMI is a sequence of 27 geometric figures that are to be copied by the examinee using paper and pencil. The figures, which are arranged in a developmental sequence, are presented on a page in a booklet above a space where the examinee is to draw an identical figure. The Visual Perceptual supplemental test uses the same geometric figures but, rather than drawing, requires the examinee to select by pointing to the geometric figure that is the same as a model. The Motor Coordination supplemental test requires the child to trace the same stimulus forms with a pencil without going outside double-lined paths.

The BOTMP consists of eight subtests, three fine motor tests, four gross motor tests, and one combination test, designed to provide a comprehensive index of motor proficiency. Normative data provide standardized scores for the appropriate age groups.

The WISC-III is a standardized measure of cognitive functioning in children. Participants were administered an abbreviated version by a master's level psychologist, including the Similarities and Vocabulary subtests of the Verbal Scale and the Block Design and Object Assembly subtests of the Performance Scale. These four subtests were selected on the basis of their high factor loadings with the Verbal, Performance, and Full Scale IQ.[21] Subtest scores were prorated to obtain estimates of Verbal, Performance, and Full Scale IQ. Examiners were trained and supervised by a licensed child psychologist.

Numerous birth and demographic characteristics collected from hospital records included gender, race, maternal age, parity, and number of prenatal visits. The child's height, weight, and head circumference were measured at the time of the visit, as well as in infancy.

In addition to tests administered to the children, mothers were asked to complete a questionnaire cataloguing family income, maternal education, and the child's placements by month since birth. In this study, the Middle Childhood Home Observation for Measurement of the Environment (HOME) Inventory,[22] which is designed to measure the quality of a child's developmental environment, was administered in an interview form during their laboratory visit. Also, maternal receptive vocabulary for the primary caregiver was measured on the Peabody Picture Vocabulary Test (PPVT)-Third Edition,[23] serving as an estimate of cognitive ability. The same measures were obtained from the biological mother and in cases when the child had been placed outside of maternal care. The PPVT was retained as a separate measure for each of the caregivers. A full IQ assessment was prohibited because of time constraints. Finally, 114 mothers in this sample participated in an extensive drug-use interview designed to measure more precisely the amount of cocaine, alcohol, and marijuana ingested per week and the amount of tobacco smoked per day. Because this interview was instituted midway through the project, data are only available for 114 subjects (50 cocaine-exposed and 64 non-exposed).

The institutional review board of the participating hospital approved the study. Parents or guardians gave informed written consent for both their own and their child's

participation, and children provided assent for their cooperation, with confidentiality ensured for all participants.

Data Analysis

Differences between cocaine-exposed and unexposed groups were examined using χ^2 analyses for categorical variables and t tests for continuous data for both the mother and child. Stepwise multiple regressions were then used to evaluate the predictive power of cocaine exposure after controlling for confounding variables. Before inclusion in the model, relationships between possible confounding and dependent variables were examined using a correlational strategy (using a significance level of $p < .10$). This conservative level of significance was chosen to reduce the number of variables entered into the regression models while at the same time allowing analyses consistent with a teratological model to account for important demographic, environmental, and medical factors at the p less than .05 level.

Confounds examined in these analyses included maternal age, number of prenatal visits, parity at delivery, and education; family income; biological parent and caregiver PPVT-Third Edition standard scores; and HOME total score. The children's z-transformed height, weight, head circumference in infancy; current height, weight, and head circumference; age at testing; gender; and amount of time in the care of a biological parent were also considered.

Variables that correlated with outcomes were subsequently entered into multiple regression analyses hierarchically to determine whether drug effects remained after controlling for confounds. Variables were entered in the following order: current home environment; maternal prenatal characteristics, including age of biological mother, parity, number of prenatal visits, marital status, and biological mother's IQ; current caregiver's IQ; and prenatal measures of cigarette, alcohol, and marijuana exposure. Cocaine exposure status was added on the final step. Because heavily exposed children were more apt to be placed outside of maternal/family care, caregiver status was also examined in regression analyses by entering this variable after the effects of cocaine status were explored.

Cocaine was represented in a dichotomous fashion when using group (exposed vs unexposed) as a predictor in the model. In an effort to examine more closely the cocaine effect, a continuous variable of rocks per week was calculated on the basis of maternal report for the 114 in this sample for whom data were available (birth = 97; 1 year = 12; 2 years = 10). Thus, two separate sets of regressions were performed examining the effects for cocaine status for each dependent measure. Because of collinearity, if a variable did not account for a significant amount of variance when it was entered, it was eliminated from further steps of the model. However, variables that were significant when entered were kept in the model even if they became nonsignificant when another variable was entered at a later step. Race and gender were only considered as moderators provided that cocaine effects emerged. Physical mediators (weight, length, and head circumference) were also considered in instances in which cocaine effects occurred. Unless otherwise noted, only confounding mediating and moderating variables that were related at the 0.10 level will be discussed in this article.

RESULTS

Table 1 presents the sociodemographic characteristics of the entire sample. At the child's birth, biological mothers who used cocaine had higher parity, were older, experienced less prenatal care, and had less income per month than mothers who had not used cocaine. Biological mothers of cocaine-exposed children had lower receptive vocabulary scores, smoked more, drank more, and used more marijuana than mothers who had not used cocaine. Although the groups did not differ in marital status, they did differ on the custodial care variable. Fifty-six percent of the cocaine-exposed children resided outside maternal care compared with 13% of the non-exposed children. Table 2 presents the sociodemographic and anthropometric data on the children, as well as current age and 7-year-old Home Observation for Measurement of the Environment (HOME) outcomes. The group of children prenatally exposed to cocaine did not differ in race and gender, with marginal age effects (0.09) favoring cocaine-exposed children. Cocaine-exposed children did differ from non-exposed children in gestational age, infant height, weight, and head circumference, with exposed children lower on all four measures. Despite differences at birth in physical dimensions, no differences in height, weight, and head circumference were observed at 7 years of age. Finally, cocaine-exposed children received lower HOME scores at 7 years of age than their non-exposed counterparts.

Cocaine effects were also found on many of the outcome variables for the full sample. There were significant group differences on the Visual Motor Integration (VMI) Motor Coordination standard scores and Verbal and Full Scale IQ, and marginally significant differences on the fine motor subscale of the Bruininks-Oseretsky Test of Motor Proficiency (BOTMP), with the cocaine-exposed children performing less well than the non-exposed children (Table 3). Chi-square analysis indicated there were no differences between the groups in the percentage of children who attended special-education classes during either the preschool or school-age period, or significant age differences between the groups.

Correlations between potential confounding variables and outcome variables can be found in Table 4. HOME and caregiver Peabody Picture Vocabulary Test (PPVT) scores were the two variables that most frequently related to outcomes. They both correlated with the VMI, Wechsler Intelligence Scale for Children-Third Edition (WISC-III) IQ scores, and the fine motor score from the BOTMP. The biological mother's age and child's age at the time of testing both correlated negatively with all scores from the BOTMP. The number of prenatal visits was marginally related to overall VMI score and both Performance and Full Scale IQ. Cocaine correlations were the strongest of the drug correlations, relating significantly to overall VMI, Performance and Full Scale IQ, and motor performance. Correlations with the other three drugs seem to be in the motor domain when they appear at all.

Table 1. Sociodemographic Characteristics of the Biological Mother

	Exposed (n = 101)		Unexposed (n = 130)		
	Mean	SD	Mean	SD	p
Maternal					
Parity	3.1	1.7	2.0	1.9	.001
Age (yr)	28.5	4.9	22.7	5.5	.001
Prenatal visits	3.9	4.3	7.0	3.4	.001
Education (yr)	11.8	1.9	12.0	1.5	NS
Marital status (% single)	92%		84%		NS
Monthly income	$1025	$909	$1528	$978	.001
Time living with biological parent (month)	54	38	79	23	.001
Biological parent PPVT	82.1	10.5	88.1	10.2	.001
Drug history (n = 114)	n = 50		n = 64		
Cigarettes % yes	85%		19%		.001
Frequency/d	9.80	12.5	0.82	2.1	.001
Alcohol % yes	79%		25%		.001
Frequency/wk	1.87	3.1	0.08	0.2	.001
Marijuana % yes	40%		5%		.001
Frequency/wk	0.55	1.8	0.15	1.2	.05
Cocaine % yes	100%		0%		.001
Frequency rocks/wk	15.0	39.0		0	.001

PPVT, Peabody Picture Vocabulary Test; NS, not significant.

Regression analyses were conducted in the manner described previously. First, all outcome measures were presented using the dichotomous classification of group as the indication of cocaine status. Second, regressions were conducted on the subsample of 114 children (cocaine group = 50; unexposed = 64) for whom the continuous variable of number of rocks of cocaine was available. The majority of this subsample was recruited at birth (n = 92), with drug interview data for 12 children in the cohort of 1 year olds and 10 children in the cohort of 2 year olds. No differences in the demographics and outcome variables were found between the cocaine-exposed children whose mothers completed the drug interview and those whose mothers did not. In addition, the only differences obtained between cocaine-exposed children residing in maternal care and non-maternal care were in terms of gender and amount of cocaine exposure. Children residing in nonmaternal care were more heavily exposed to cocaine and more apt to be male.

As can be seen in Table 5, the dichotomous variable of cocaine status (exposed vs non-exposed) did not contribute to the prediction of any outcome variable. The most useful predictors of outcome measures in all regressions proved to be maternal vocabulary (PPVT-Revised) score and home environment at age 7 years. No significant prediction models were obtained for the VMI perceptual subscale or the gross motor subscale from the BOTMP.

All regression models remained the same in regard to the subsample involving the continuous variable of rocks of cocaine, with the exception of the overall score from both the VMI and the BOTMP. The logged value of number of

Table 2. Child Sociodemographic and Anthropometric Characteristics for the Total Sample

Measures	Exposed (n = 101)		Unexposed (n = 130)		
	%/Mean	SD	%/Mean	SD	p
Current age (yr)	7.32	0.46	7.20	0.56	NS
Race (African-American)	99%		95%		NS
Gender (male)	54%		56%		NS
Gestational age	37.7	2.27	38.6	2.27	.01
Infant height (z score)	−1.35	0.8	−0.72	1.0	.001
Infant weight (z score)	−1.01	1.0	−0.41	1.0	.001
Infant head circumference (z score)	−1.32	0.8	−1.00	0.8	.001
7-yr height (cm)	124.4	5.6	124.7	6.1	NS
7-yr weight (kg)	29.5	9.4	29.4	8.2	NS
7-yr head circumference (cm)	51.6	1.9	51.7	1.8	NS
7-yr HOME total	38.9	7.6	41.8	7.7	.01

HOME, Home Observation for Measurement of the Environment; NS, not significant.

Table 3. Seven-Year Outcomes by Cocaine Status

	Exposed (n = 101)		Unexposed (n = 126)		
	Mean	SD	Mean	SD	p
VMI SS	92.18	11.43	94.26	12.67	NS
VMI Motor SS	84.29	10.13	87.43	10.31	.02
VMI Visual SS	88.89	14.09	89.50	16.36	NS
WISC-III Verbal IQ[a]	84.52	14.19	88.55	14.62	.03
WISC-III Performance IQ[a]	82.95	14.76	85.33	15.69	NS
WISC-III Full Scale IQ[a]	81.07	14.09	84.92	15.65	.05
BOTMP Fine SS	42.75	9.47	45.05	10.88	NS
BOTMP Gross SS	47.60	10.71	49.21	9.74	NS
BOTMP Total SS	44.81	11.01	47.19	10.48	NS
Preschool special education	15%		12%		NS
Elementary special education	2%		2%		NS

VMI SS, Developmental Test of Visual-Motor Integration, Fourth Edition Standard Score; WISC-III, Wechsler Intelligence Scale for Children-Third Edition; BOTMP SS, Bruininks-Oseretsky Test of Motor Proficiency Standard Score; NS, not significant.
[a]Abbreviated version of the WISC-III based on four subtests.

rocks of cocaine added significantly to the prediction of VMI total (confidence interval −4.06−−0.18) and marginally to BOTMP (confidence interval −3.314–0.174). Thus, in addition to maternal vocabulary and home environment, the amount of cocaine ingested during the fetal period is an important predictor of motor and visual motor integration skills.

DISCUSSION

Results indicate that, at age 7 years, children who were exposed prenatally to cocaine continue to display deficits on tasks of verbal, visual motor integration, and fine motor skills. The cocaine effect, however, was rendered nonsignificant by inclusion of sociodemographic and environmental variables, especially the caregiver's vocabulary and the child's home environment at the time of testing. These results indicate that prenatal exposure to cocaine, per se, may overlap with other cognitive risk factors experienced by children of low-income families. The amount of cocaine exposure, however, did predict lower visual motor and motor skills beyond the environmental factors for a subset of children for whom quantitative exposure data were measured. In a separate cohort, our research group has found cognitive differences associated with cocaine exposure in children aged 2 years.[12] The larger sample size and more specific quantitative assessment of drug exposure in that cohort may have allowed for more sensitive detection of differences between exposure groups. Nonetheless, environmental factors impact motor outcomes as well.

This study has several limitations. Although cocaine exposure was determined through a careful review of hospital records, including urine screens and maternal report,

Table 4. Seven-Year Outcomes Correlated with Birth Data

	VMI	Visual Perception	Motor Coordination	WISC Verbal	WISC Performance	WISC Full Scale	Gross Motor	Fine Motor	Motor Total
Test age	−0.08	−0.04	−0.07	0.00	−0.00	−0.00	−0.05	−0.14**	−0.11*
Weight (z score)	0.02	0.12*	0.05	0.15**	0.09	0.13**	−0.03	−0.04	0.01
Height (z score)	0.05	0.04	0.01	0.08	0.08	0.09	−0.03	−0.01	−0.01
Head circumference (z score)	0.12*	0.07	0.01	0.08	0.07	0.09	−0.01	0.10	0.02
Maternal age[a]	−0.02	−0.01	−0.07	−0.01	−0.05	−0.04	−0.14**	−0.24***	−0.18***
Maternal education[a]	0.20**	0.11	0.08	0.22**	0.14*	0.21**	−0.08	0.13	0.01
Parity	0.15**	−0.10	−0.07	−0.19**	−0.19**	−0.21***	0.01	−0.11*	−0.06
Prenatal visit	0.13*	−0.03	0.10	0.11	0.12*	0.13*	−0.01	0.16**	0.10
Family income	−0.05	0.09***	0.07	0.16**	0.12	0.16**	0.07	0.06	0.09
Time w/ parent	0.04	−0.03	−0.06	−0.01	−0.03	0.02	−0.02	0.02	0.01
Maternal PPVT[a]	0.27†	0.20***	0.21***	0.27†	0.33†	0.33†	−0.02	0.19***	0.09
Home total	0.14**	0.07	0.20***	0.34†	0.17**	0.29†	0.11	0.18***	0.16**
Gender	0.15**	−0.03	−0.04	0.08	0.14**	0.13**	0.22***	0.06	0.22***
Cocaine[b]	−0.26***	−0.08	−0.08	−0.18*	−0.19**	−0.20**	−0.15	−0.32†	−0.26***
Tobacco[b]	−0.19**	−0.07	−0.09	−0.09	−0.13	−0.12	−0.03	−0.16*	−0.11
Alcohol[b]	−0.15	−0.06	−0.06	−0.05	−0.12	−0.10	0.06	−0.25***	−0.10
Marijuana[b]	−0.13	0.02	0.09	0.03	−0.14	−0.06	−0.13	−0.11	−0.15*

PPVT, Peabody Picture Vocabulary Test; VMI, Developmental Test of Visual-Motor Integration; WISC, Wechsler Intelligence Scale for Children.
[a]Maternal refers to the biological mother.
[b]All drug correlations are based on the subsample of 114 children with drug interviews.
*p < .10, **p < .05, ***p < .01, †p < .001.

Table 5. Final Regression Models Using the Dichotomous and Continuous Cocaine Variable

	Parameter Estimate Group	SE	p Value	Parameter Estimate Crack	SE	p Value
VMI						
HOME	0.09	0.12	.42	0.04	0.15	.81
PPVT-R	0.34	0.10	.001	0.24	0.12	.04
Group/crack	0.09	1.79	.96	−2.12	0.99	.03
Motor Coordination						
HOME	0.18	0.10	.09			
PPVT-R	0.21	0.08	.01			
Group	−0.75	1.59	.64			
WISC Verbal IQ						
HOME	0.46	0.14	.001			
Parity	−1.19	0.55	.03			
PPVT-R	0.36	0.11	.02			
Group	0.46	2.21	.83			
WISC Performance IQ						
HOME	0.15	0.15	.33			
Marital status	5.17	3.34	.12			
PPVT-R	0.43	0.12	.001			
Group	1.03	2.29	.65			
WISC Full Scale IQ						
HOME	0.32	0.15	.03			
Parity	−1.41	0.57	.01			
Marital status	5.77	3.37	.09			
PPVT-R	0.41	0.12	.001			
Group	1.25	2.32	.59			
Fine Motor						
HOME	0.17	0.10	.01			
Prenatal visits	0.28	0.19	.15			
PPVT-R	0.21	0.08	.01			
Group	0.11	1.62	.95			
Total Motor						
HOME	0.20	0.09	.07	0.22	0.12	.08
Maternal age	−0.26	0.14	.06	−0.01	0.17	.95
Group/crack	−0.24	1.65	.88	−1.57	0.89	.08

HOME, Home Observation for Measurement of the Environment; PPVT-R, Peabody Picture Vocabulary Test-Revised; WISC, Wechsler Intelligence Scale for Children; VMI, Developmental Test of Visual-Motor Integration.

misclassification of drug exposure is possible. Further, as previously reported,[9] the groups were initially recruited at different ages, ranging from birth to 2 years old. Although there were no differences in outcomes between the recruitment cohorts, and age at recruitment did not account for a significant amount of variance in any of the models, there is still the possibility that the accuracy of maternal report of drug use may be less reliable the further removed from the pregnancy experience. In addition, although the cocaine-exposed group was distinguished from the unexposed group by exposure to cocaine in utero, both groups frequently used other drugs, especially alcohol and nicotine.

The finding that little of the variance in developmental outcomes measured in this study was accounted for by family income and none by maternal education was somewhat unexpected, but was likely because of a restricted socio-demographic range in the sample. The strong relationship between developmental outcomes at 7 years and both the home environment and maternal vocabulary skills provides evidence for the influence of environmental factors in children's development.

Both cocaine-exposed and unexposed children participating in this study did poorly when compared with the normative standards on all facets of assessment, but particularly on the Wechsler Intelligence Scale for Children-Third Edition (WISC-III). These results are comparable to other studies suggesting that children from low-income, inner-city families score below average in development.[24–26] The current findings were also consistent with other studies suggesting that children from low-income families, both those involved and not involved with drug use, show progressive lags in development.[14,27–28] Regression analyses using a subsample of children with continuous data on cocaine exposure tentatively suggested that prenatal drug exposure represents an added developmental risk beyond those encountered by children raised in an impoverished environment, at least for visual and motor skills. This additional risk may arise from a lack of suitable and consistent caregiving associated with a drug-using environment or may represent a limited ability by the child to adapt to environmental challenges. This is consistent with the literature on the adverse effects of poverty on child development. A more precise measurement of the amount of cocaine exposure would enhance our ability to make a definitive statement regarding this matter.

In this study, dichotomous classification of cocaine status failed to be a useful predictor of developmental outcome, whereas severity of cocaine exposure predicted poorer visual motor and motor skills for the subgroup on whom this information was available. Two possible explanations for this effect can be offered. First, the dichotomous classification (exposed/non-exposed) may mask important differences in the groups. Alternatively, it may be because during the course of 7 years since the time of the prenatal exposure, the postnatal environmental variables have had a strong influence on development that masks subtle prenatal drug-exposure effects.

Because a sequential cohort recruitment schedule did not allow reliable assessment of the severity of exposure for a subgroup, there was limited information collected on severity of exposure. Children in the cocaine-exposed group therefore may have been exposed to a smaller amount of the drug than would be required to produce a strong effect. Future studies should document more precisely the amount and timing of cocaine exposure.

Most of the women who used cocaine also smoked cigarettes and drank alcohol during their pregnancy, as did some of the women who did not use cocaine. Because effects on the outcomes were small in the present study, partialing them out across multiple drugs would obscure the overall finding that, in general, children with poorer home environments and whose mothers have poorer vocabularies are performing poorly across all developmental outcomes. The clinical and policy implications from this study are clear. To lessen biological risks and enhance development, prenatal programs that provide drug treatment to women, especially in disadvantaged families, should, in addition to treating drug use, include interventions designed to improve both the mothers' academic and caregiving skills.

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