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Motor Development of Cocaine-exposed Children at Age Two Years

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Abstract

Objective—This article was designed to investigate effects of prenatal cocaine exposure on motor development of young children from a predominately underprivileged, urban population.

Methodology—A total of 260 infants and young children were initially recruited from either the newborn nursery or the at-risk pediatric clinic of an urban teaching hospital. Prenatal history and birth outcomes were collected from medical records. Demographic characteristics and additional drug histories were obtained from the mothers. The 199 subjects (98 cocaine-exposed and 101 unexposed) who returned at age 2 years were assessed by examiners blinded to drug exposure status using the Peabody Developmental Motor Scales.

Results—Compared with control subjects, the cocaine-exposed group performed significantly less well on both the fine and the gross motor development indices. Mean scores for both groups were within the average range on the gross motor index, but greater than 1 standard deviation below average on the fine motor index. Differences were significant on the balance and the receipt and propulsion subscales of the gross motor scale, and on the hand use and the eye–hand coordination subscales of the fine motor scale. Cocaine status independently predicted poorer hand use and eye–hand coordination scores. There also was an effect of alcohol exposure on the receipt and propulsion subscale.

Conclusions—Findings indicate that deficiencies in motor development remain detectable at 2 years of age in children exposed to drugs prenatally. Although other environmental variables may influence motor development, children exposed to cocaine and to alcohol in utero may encounter developmental challenges that impede later achievement.

Although the recent epidemic of cocaine use may have subsided,[1,2] concerns about the development of many children exposed to cocaine in utero remain relevant. There are several possible mechanisms by which cocaine may damage the developing nervous system of the human fetus,[3] suggesting that neural functions associated with movement, such as those regulated by the hypothalamic and extrapyramidal systems, should be examined closely. Specific findings demonstrating a strong and explicit effect of fetal cocaine

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exposure on infant development, however, have been difficult to confirm, in part because of numerous confounding factors.[4] In particular, many studies had small sample sizes and/or lacked control for the polydrug exposure that is commonly associated with cocaine exposure. Additionally, previous research frequently failed to consider other factors, such as amount of prenatal care, maternal education, and prematurity, that also have been shown to affect developmental outcome.[5,6]

Numerous studies have focused on the neurobehavioral sequelae of prenatal cocaine exposure on newborns or young infants, many of which reported significant negative effects of cocaine exposure. Reviews of this literature, however, note several inconsistencies in the nature and extent of the deficits.[7,8] Animal studies of neurobehavioral development in rodents exposed to cocaine found few major dysmorphic effects, but report changes in startle and locomotor activity, suggesting specific rather than global effects on the motor system.[9,10]

Results of an early study found a relationship between prenatal exposure to cocaine and performance on the motor cluster of the Brazelton Neonatal Behavioral Assessment Scale (BNBAS),[11] but later findings have been mixed. Most results demonstrated either no cocaine effect,[12,13] or an effect in an area other than the motor domain.[14–17] One study[18] investigating the effects of prenatal exposure to several drugs reported that the duration of alcohol and marijuana exposure predicted BNBAS motor behavior at age 2 days, and duration of cocaine exposure predicted motor behavior at 28 days. Another study[19] found that although there were no differences at 1 to 3 days of age on the BNBAS, the motor cluster was the only domain of the BNBAS on which cocaine-exposed neonates did less well at days 11 to 30 than did a no-drug comparison group. A recent, well-controlled study[20] reported that maternal use of cocaine in the second and third trimesters was associated with poorer infant motor maturity and tone. Meconium cocaine concentration also has been found to have a negative dose–response relationship to age-appropriate motor and regulation-of-state behaviors.[21]

Several studies have investigated motor development in cocaine-exposed children beyond the neonatal period.[22,23] One well-controlled study[24] reported that a greater proportion of the cocaine-exposed group displayed an at-risk level of performance on both the Movement Assessment of Infants and the Alberta Infant Motor Scale at 4 months and had lower motor scores at 7 months than did a matched control group. In a study of high-risk infants,[25] 41% of cocaine-exposed infants exhibited hypertonia at age 6 months, which resolved in most children by age 24 months. Finally, two separate studies using the Psychomotor Developmental Index Score from the Bayley Scales of Infant Development found poorer performance by children who were exposed to cocaine prenatally at 4 and 17 months,[26,27] whereas others found no group differences.[28,29]

The current study investigated the hypothesis that 2-year-olds exposed to cocaine in utero would perform less well on a standardized assessment of fine and gross motor skills compared with a group of unexposed children recruited from the same high-risk population. Specific hypotheses include the following: 1) cocaine-exposed children will perform less well than comparable unexposed children at 2 years of age on standardized measures of

motor development; and 2) cocaine exposure will account for a significant proportion of the variance in motor outcomes, independent of other confounding variables.

METHOD

Subjects

Participants included cocaine-exposed infants and a comparison group of unexposed infants recruited from the same population of minority urban poor. Most (97%) of mothers were African-American, and most (96%) were receiving Medicaid and living in the inner city.

Mothers and infants were recruited prospectively over a 1-year period either from the newborn nursery at the time of delivery or at a 1- or 2-year well-baby visit in an ambulatory pediatric clinic at a private, university-affiliated urban hospital as part of a longitudinal study.[26] The clinic serves primarily an inner city population of infants and children who are at increased risk of developmental and medical problems associated with poverty, including poor prenatal care, poor nutrition, and neglect, as well as with prenatal drug exposure.

Drug exposure was determined by a combination of medical chart review, maternal and/or infant urine toxicology results, and/or clinical interviews. Urine samples were obtained in the prenatal clinic or at the time of delivery through the hospital screening protocol based on the after-risk factors: lack of adequate prenatal care, precipitous delivery, history of drug use, self-reported drug use, previous involvement with the Department of Human Services concerning abuse/neglect of children, intoxication apparent to hospital staff, or impaired cognitive/emotional functioning. Samples were analyzed by enzyme immunoassay using the Syva Emit method (Syva Company, Palo Alto, CA), with assays performed for cocaine, barbiturates, amphetamines, marijuana, and heroin. The specificity of this measure for benzoylecgonine, the most common metabolite of cocaine in adults, is 99% at a concentration of 300 ng/mL. Follow-up thin-layer chromatography or gas chromatography was performed for confirmation. In a separate clinical study conducted previously at the same maternity hospital, this combination of clinical indications, historical information, and voluntary urine testing identified 95% of cocaine-positive deliveries.

Infants were excluded if the mother was younger than age 17 years or if the infant weighed ≤1500 g at birth. All mothers whose records or interview identified primary psychiatric problems or low intellectual status; positive HIV status; or positive drug test results for PCP, amphetamines, barbiturates, or heroin were excluded. Women who used alcohol, tobacco, or marijuana during pregnancy were retained in both groups. Of the 260 infants enrolled initially, 199 (76%) (98 cocaine-exposed and 101 unexposed) completed the 2-year assessment. The study was approved by the institutional review board of the participating hospital, and written informed consent was obtained for all participants.

Measures

Maternal and infant demographic and medical characteristics were collected from medical records. These included maternal age, race, gravidity, number of prenatal visits, and type of medical insurance. Infant characteristics included gender, 5-minute APGAR score, gestational age, length, weight, and head circumference.

The Maternal Postpartum Drug Interview[30] was administered to mothers whose infants were recruited at birth to quantify maternal drug use. This extensive interview was independent of any other evaluations of drug history conducted in the hospital for clinical purposes. Of these women, 89 (43 cocaine-positive, 46 cocaine-negative) returned for the 2-year visit, and those data were used in the present analyses as a representative subsample of the entire group.

Mothers were asked to recall the amount and frequency of drug use per day for the month before conception and for each trimester of pregnancy. For tobacco use, mothers were asked to recall the number of cigarettes (joints) smoked per day. Frequency (number of days of use per week) for alcohol, marijuana, and cocaine use was recorded for the same period. The number of marijuana cigarettes and the number of drinks of beer, wine, or hard liquor consumed per day, with each drink equivalent to .5 oz of absolute alcohol, also were recorded. For cocaine, the number of "rocks" and the amount of money spent also was computed. The frequency of use then was multiplied by the amount used per day to compute a severity of use score. Scores from 1 month before and the trimesters then were averaged for a mean total use score over the entire pregnancy for each drug. For inclusion in regression analyses, scores were subjected to a log (× + 1) transform to normalize distribution.

The Peabody Developmental Motor Scales (PDMS)[31] was administered to all children within 1 month of their second birthday by qualified examiners blinded to the children's drug-exposure status. The PDMS is a standardized measure of gross and fine motor abilities that provides developmental motor quotient (DMQ) scores for children from birth to 83 months. It is a stable assessment tool that has been used in previous studies of motor development in drug-exposed young children.[24]

As administered to the 2-year-olds in this study, the gross motor scale consists of four subscales that measure large muscle activities: balance, locomotor, nonlocomotor, and receipt and propulsion of objects. The fine motor scale consists of three subscales that measure precise movement of the small muscles: hand use, eye-hand coordination, and manual dexterity. Tasks are scored on a 3-point scoring system: 0 credit for unable; 1 point for partially capable; and 2 points for meets criterion completely. The test is norm-referenced, and subscale and total scores yield developmental quotients that have a mean of 100 and a standard deviation [SD] of 15.

Data Analysis

Group Differences—Groups were compared on demographic characteristics using Wilcoxon rank sum tests for continuous data and χ^2 analyses for categoric variables. Separate multivariate analyses of variance, using the four subscales of the gross motor or the three subscales of the fine motor portions as dependent variables, and drug status as the independent variable, were used to investigate group differences. Separate analyses of variance were conducted on gross and fine DMQ total scores. Similarly, group differences on each of the four subscales of the gross motor scale and the three subscales of the fine motor scale also were examined.

Confounding Variables and Multivariate Analyses—The following variables were considered as possible confounders of outcomes: infant race, gender, and 5-minute APGAR score; maternal number of prenatal care visits, marital status, age at delivery, and educational level; and family income and number of persons living in the home. For those outcomes for which significant group differences were found, a check of relationships between possible confounding variables and motor outcomes was conducted, using a correlation at the $P < .1$ significance level as the criterion.[32]

To test the relative influence of confounders, including other drugs, versus cocaine exposure on outcomes, a series of hierarchical regression analyses were performed on those variables for which group differences were identified. Gross and fine motor total and subscale scores were used as the dependent variables. For each outcome, the confounding demographic variables, as described above, were entered on the initial steps. These were followed by severity of cigarette, alcohol, and/or marijuana use when those drugs were identified as correlates of the outcome considered. Finally, cocaine exposure, coded as exposed or unexposed, was entered.

Birth outcomes including gestational age, birth weight, length, and head circumference were also examined as possible mediators, because they have been shown in previous studies to be related to drug exposure[6,15,33] and to be related to later developmental outcomes. On those analyses in which a significant drug effect was found, the regressions were conducted again, with each of the potential mediators entered before the drug variable.

RESULTS

Differences in demographic variables are shown in Table 1. There were no significant group differences in maternal ethnicity or marital status. Mothers who used cocaine were older and had fewer years of education, higher parity, and less prenatal care. Cocaine-exposed children were more likely to be female and to have a lower gestational age and reduced birth length, birth weight, and head circumference, even after adjustment for gestational age.

Although a greater percentage of cocaine-exposed infants than unexposed infants from the original 261 newborns recruited (84% vs 70% of the unexposed comparison infants) returned for the 2-year follow-up, there were no significant differences between the group seen at the 2-year visit versus the group not seen in regard to gender, gestational age, 5-minute APGAR score, or birth outcomes, including length, weight, and head circumference. There were also no differences in maternal age, parity, or number of prenatal visits between the mothers whose babies were seen at 2 years and those who missed the visit. Mothers who had their children tested at 2 years, however, did smoke significantly more cigarettes during their pregnancy than those who did not come in for the visit. This is likely related to the higher rate of retention for the cocaine-using women and the high rate of tobacco use in that group (Table 2).

Comparing the 45% of mothers who received the postpartum drug interview versus those who did not (89 of 199), there were no significant differences in the percentage of mothers who used cocaine, nor were there significant differences in any of the maternal demographic

characteristics. Additionally, there were no significant differences in child demographic or birth outcomes between those 2-year-olds whose mothers received the postpartum interview versus those whose mothers did not.

Severity of Drug Exposure and Outcome

Data obtained from the postpartum interview indicated that mothers who used cocaine also smoked more cigarettes per day and that their severity of alcohol and marijuana use during pregnancy was greater (Table 2). These results are consistent with previously reported patterns of polydrug use among pregnant women who use cocaine.[6,34]

Motor Scores

Exposed and unexposed groups did not differ on age at testing. Multivariate analysis revealed significant group differences in the attainment of both gross ($F_{(4,194)} = 6.5$; $P < .001$) and fine ($F_{(3,195)} = 5.3$; $P < .001$) motor skills. Total scores on both the fine and gross motor scales revealed poorer performance by the cocaine-exposed group in overall ability (Table 3). The average total score for both groups was below the mean on the gross motor scale and >1 SD below the normative group on the fine motor scale. Using a cutoff score of 80 as recommended in the PDMS manual, 15% of the cocaine-exposed group versus 7% ($\chi^2 = 3.46$; $P = .06$) of the unexposed group was classified as at-risk on the gross motor scale, whereas 34% of the cocaine-exposed children versus 21% ($\chi^2 = 3.84$; $P = .05$) of the unexposed were at risk on the fine motor scale.

Cocaine-exposed infants did significantly less well on the balance and the receipt and propulsion sub-scales of the gross motor scale and the hand use and the eye-hand coordination subscales of the fine motor scale (Table 3).

Confounding Variables

Maternal age at birth, number of prenatal visits, and maternal education correlated with both the gross motor and the fine motor development quotient scores (Table 4). Parity and infant gender did not correlate with either total score. On the gross motor subscales, the balance and the receipt and propulsion scores correlated with maternal age and number of visits. Maternal education correlated with balance, and gender correlated with receipt and propulsion. On the fine motor subscales, maternal age was the only significant correlate of hand dexterity, and none of the demographic confounders correlated with eye-hand coordination.

Total severity of alcohol use was associated inversely with the total ($r = -.22$), the balance ($r = -.21$), and the receipt and propulsion ($r = -.22$) scores of the gross motor scale. Overall severity of cigarette use was associated negatively with the total ($r = .18$) and the balance ($r = -.27$) scores of the gross motor scale and the eye–hand coordination ($r = -.22$) scores of the fine motor scale. Severity of marijuana exposure did not correlate with any motor outcome.

When timing during pregnancy was considered, severity of alcohol use in the month before conception and in the first trimester correlated with gross motor total score, severity of

cocaine use in the first trimester correlated with both gross and fine motor total scores, and severity of tobacco use in the second and third trimester showed a trend with both fine and gross motor total scores (Table 4).

Mediating Variables

Gestational age, birth weight, and birth length correlated with gross motor total DMQ (Table 4). Gestational age, birth weight, birth length, and head circumference correlated with fine motor total DMQ. On the gross motor subscales, gestational age and birth length correlated both with balance and with receipt and propulsion. Additionally, head circumference and birth weight correlated with receipt and propulsion. On the fine motor subscales, gestational age, birth length, and head circumference correlated both with eye-hand coordination and with hand dexterity. Additionally, birth length correlated with eye-hand coordination.

Independent Effects

Results of hierarchical regression on the fine motor subscales indicated that after controlling for all potential confounders, cocaine group status predicted hand use subscale scores and eye-hand coordination subscale scores (Table 5). The effect of cocaine on these subscale scores remained significant after the potentially mediating birth outcome variables were considered.

For the gross motor outcomes, the severity of alcohol use in the month before pregnancy was a significant predictor of the receipt and propulsion sub-scale score (Table 5). Again, the effect of alcohol remained significant after the potentially mediating birth outcome variables were entered into the regression equation.

DISCUSSION

Two-year-old children who had been exposed prenatally to cocaine performed less well on a standardized test of motor development in comparison with a group of unexposed children of similar race, age, and socioeconomic status. Cocaine use during pregnancy had the strongest relationship, with two fine motor skills, hand use and eye-hand coordination. Receipt and propulsion skill was best accounted for by severity of alcohol exposure.

These findings indicate a lag in motor development beyond the neonatal period in children who were exposed prenatally to cocaine. An earlier report of transient motor disorders[25] that resolved beyond the age of 1 year may have lacked the power to detect these differences, because of the type of measure used. Lack of differences between cocaine-exposed and unexposed children on the PDMS reported by other investigators[24] may have been attributable to the small number of participants in the earlier study or to the poor performance of both groups on the PDMS.

Although most scores in the present study fell within the normal range on the gross motor scale, the proportion of cocaine-exposed children whose scores indicated an elevated level of risk was double that of the unexposed group. In addition, although the mean score was in the delayed range for both groups, there was evidence for an increased rate of abnormal fine motor development in greater than one third of the cocaine-exposed group.

The findings also indicate that maternal age and number of prenatal visits accounted for a significant amount of the variation in motor development among the study participants. Gross motor and fine motor developmental quotients, as well as the hand use and the receipt and propulsion subscale scores, were significantly correlated with maternal age. Number of prenatal visits correlated significantly with fine motor total scores and showed a trend in relationship to gross motor total scores and receipt and propulsion subscale scores. The only effect of gender detected was on the receipt and propulsion subscale. This reinforces previous studies[35] and current theory[36] that motor development is a product of the interaction of genetic attributes, biologic maturation, and environmental stimulation. Maternal age >30 has been linked to poorer motor development in children whose mothers used alcohol during pregnancy.[32]

Alternatively, the relationships may reflect the confounding of maternal age with drug exposure in this sample. When the correlation between maternal age and motor outcomes was calculated separately for the cocaine-exposed and unexposed groups, the relationships all failed to reach significance. In the present study, the mean age of the mothers in either group was neither exceptionally old nor exceptionally young, suggesting that the relationship found between maternal age and motor outcomes may be attributable to the fact that older mothers used more drugs.

The relationship between prenatal alcohol exposure and gross motor development found in this report is consistent with that for other motor deficiencies reported in children with fetal alcohol syndrome.[37] Motor deficiencies, which often are associated with central nervous system pathology,[38] are likely to persist to later ages.

Because motor skills are an integral component of early school-age skills such as writing, music, art, and sports, it is important to diagnose and treat early problems in the motor domain. Motor control difficulties also may be related to later problems such as attention deficit, hyperactivity, or learning disorders.

There are several limitations to the present study. The most important caveat is that the findings do not establish a causal relationship between prenatal cocaine exposure and later developmental delays. Although motor development, compared with language or social skills, appears to have a greater neurobiologic basis and may be relatively less affected by cultural factors[39–42], the environment in which the children are raised will influence the rate and level of motor development. This point is most evident in the relatively low correlations between birth and maternal variables and motor outcomes. Practitioners, as well as researchers, must consider postnatal factors, particularly the adequacy of the home environment and maternal IQ and psychological status, in their evaluation and treatment of children who have a history of prenatal drug exposure.

It also is possible for children living in a household where cocaine is present to be exposed to the drug postnatally.[43,44] For example, in a separate ongoing study involving a different but demographically similar group of mothers from the same inner city population, drug use data were collected at birth and at 6 months. Of those who were using cocaine at the time of

delivery, 80% returned for the 6-month follow-up. Of this group, which might be considered a conservative estimate, 61% still were using cocaine (L. Singer, unpublished data, 1998).

Although assessments were conducted by examiners masked to drug exposure status, infants were tested with their caregivers present and it may have been possible to identify exposure status of some infants from the caregiver's characteristics or behaviors. The possibility of examiner bias, therefore, cannot be ruled out.

Finally, presence or absence of cocaine exposure was established through a review of medical records that included urine drug testing and interviews, but quantification of cocaine use and other drug use was based solely on maternal report, which may be unreliable.[45] Although the lack of stricter drug use detection procedures may have allowed mothers who used cocaine into the control group and weakened our power to detect difference, this lack of power would not negate the differences we did find.

Pediatricians should keep a balanced perspective when working with children who were exposed to cocaine prenatally. As a risk factor for atypical motor development, prenatal drug exposure is a marker for many other potential problems that can have strong negative impact on a child's development. Some of the factors associated with prenatal drug exposure, including increased risk for neglect and abuse,[46] parental psychological disorders,[27] and SIDS,[47] also can affect development adversely.

From a practical viewpoint, although lags in motor skill attainment associated with in utero exposure to drugs may not have resolved by age 2 years, most are within the scope of developmental problems seen in children during the normal course of clinical practice. The literature to date suggests that children who were exposed to cocaine and other drugs prenatally will benefit from intervention techniques, such as physical, occupational, and speech therapy, as much as any other child with similar motor problems.[48] The critical difference when working with drug-exposed children is that the social/environmental factors associated with drug exposure require the pediatrician to maintain continued, diligent observation of the child's behavioral and cognitive, as well as physical, development.

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ABBREVIATIONS

BNBAS	Brazelton Neonatal Behavioral Assessment Scale
PDMS	Peabody Developmental Motor Scales
DMQ	developmental motor quotient
SD	standard deviation

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TABLE 1

Demographics

	Exposed (n = 98)	Unexposed (n = 101)	Rank Sum, χ^2, or ANCOVA Test (df)
Mother			
Age (y)	28.2 (4.8)	22.9 (5.1)	$z = 7.05^{***}$
Parity	2.6 (1.9)	1.7 (1.1)	$z = 3.79^{***}$
Prenatal visits	4.2 (4.3)	6.9 (3.3)	$z = -4.9^{***}$
Education (y)	11.9 (1.7)	12.4 (1.6)	$z = -2.0^{*}$
Infant			
Gender (% female)	57%	43%	$\chi^2 = 4.5^{*}$
Gestational age (weeks)	37.8 (2.2)	38.8 (2.2)	$z = -3.3^{**}$
5-Minute Apgar	8.7 (0.7)	8.7 (1.1)	$z = -0.13$)
Head circumference[a] (cm)	32.8 (0.1)	33.4 (0.1)	$F_{(1,171)} = 6.6^{***}$
Birth weight[a] (g)	2863 (42)	3102 (41)	$F_{(1,175)} = 16.0^{***}$
Birth length[a] (cm)	47.4 (0.2)	48.7 (0.2)	$F_{(1,172)} = 12.5^{***}$

[a]Gestational age covaried.

*$P < .05$;

**$P < .01$;

***$P < .001$.

TABLE 2

Maternal Drug Use During Pregnancy

Variable	Exposed (*n* = 43) Mean (SD)	Unexposed (*n* = 46) Mean (SD)	Wilcoxon Rank Sums
Cigarettes/day	10.0 (12.1)	1.2 (3.3)	$z = 6.19^{****}$
Alcohol oz/wk	2.1 (3.6)	0.1 (0.3)	$z = 5.00^{****}$
Marijuana cigarettes/wk	0.6 (1.9)	0.1 (1.2)	$z = 4.21^{****}$

$^{****}P < .0001$.

TABLE 3

Peabody Developmental Motor Scales

	Exposed (*n* = 98) Mean (SD)	**Unexposed (*n* = 101) Mean (SD)**	***P* Value**
Age adjusted for prematurity (mo)	24.4 (0.4)	24.3 (0.5)	NS
Fine motor			
Hand use	84.9 (8.0)	88.9 (8.7)	.001
Eye-hand coordination	76.9 (11.7)	81.5 (9.8)	.01
Manual dexterity	85.9 (7.5)	87.5 (7.9)	NS
Total DMQ[a]	79.9 (7.9)	84.7 (10.5)	.001
Gross motor			
Balance	85.5 (13.4)	90.6 (12.5)	.01
Nonlocomotor	89.5 (13.8)	88.4 (16.2)	NS
Locomotor	90.9 (11.4)	92.1 (11.1)	NS
Receipt and propulsion [*]	93.8 (10.7)	98.3 (6.3)	.001
Total DMQ[a]	89.7 (11.0)	93.1 (8.9)	.05

[*] Separate rather than pooled variance used.

TABLE 4

Correlations Between Confounder/Mediator Variables and Motor Outcomes

	Gross Motor DMQ	Fine Motor DMQ
Maternal age	−0.16*	−0.14*
Prenatal visits	0.14#	0.16*
Maternal education	0.14#	0.13#
Cigarettes†		
Month before	−0.15	−0.12
1st Trimester	−0.13	−0.16
2nd Trimester	−0.18#	−0.20#
3rd Trimester	−0.18#	−0.18#
Total	−0.18#	−0.17
Alcohol‡		
Month before	−0.27**	−0.11
1st Trimester	−0.24*	−0.12
2nd Trimester	−0.14	−0.04
3rd Trimester	−0.13	−0.06
Total	−0.22*	−0.10
Marijuana§		
Month before	−0.00	−0.07
1st Trimester	−0.04	−0.08
2nd Trimester	−0.02	−0.06
3rd Trimester	0.02	−0.05
Total	−0.01	−0.07
Cocaine‖		
Month before	−0.16	−0.20#
1st Trimester	−0.24*	−0.24*
2nd Trimester	−0.12	−0.18
3rd Trimester	−0.14	−0.13
Total	−0.23*	−0.23*
Gestational age	0.14*	0.16*
Birth weight	0.17*	0.19**
Birth length	0.16*	0.16*
Head circumference	0.06	0.16*

#P < .10;

*P < .05;

**P < .01.

† Number of cigarettes per day.

‡ Number of ounces absolute alcohol per week.

§ Number of marijuana cigarettes per week.

// Number of rocks per week.

TABLE 5

Hierarchical Regression Analyses

Variable	Beta (SE)	R^2	*P*
Fine motor			
Criterion: eye-hand coordination			
Step 1: cigarettes/day	−1.69 (0.98)	0.03	.09
Step 2: cocaine group	7.90 (3.54)	0.14	.01
Criterion: hand use			
Step 1: maternal age	−0.20 (0.11)	0.02	.07
Step 2: gender	−3.15 (1.24)	0.05	.01
Step 3: cocaine group	4.12 (1.39)	0.09	.001
Gross motor			
Criterion: receipt and propulsion			
Step 1: maternal age	−0.33 (0.12)	0.04	.01
Step 2: maternal education	0.33 (0.39)	0.05	NS
Step 3: number of visits	0.31 (0.17)	0.06	.06
Step 4: gender	3.62 (1.31)	0.10	.01
Step 5: alcohol/month before	−2.87 (0.99)	0.22	.01
Step 6: cocaine group	−0.79 (2.20)	0.22	NS