



Developmental outcomes and environmental correlates of very low birthweight, cocaine-exposed infants

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Abstract

Fetal cocaine exposure may have differentially adverse effects on developmental outcomes of very low birthweight (VLBW) infants. As part of a longitudinal study, 31 cocaine-positive very low birthweight infants, and age, race and socioeconomic status matched VLBW controls enrolled at birth were followed. Neonatal maternal–child interactions, concurrent maternal psychological characteristics and environmental factors conceptualized as important for child outcome were assessed as well as standard developmental outcomes at 3 years.

In the neonatal period, cocaine-exposed VLBW infants who remained in maternal custody tended to be rated as less responsive and their mothers as less nurturing, less emotionally available and with a tendency to use more maladaptive coping mechanisms than nonexposed VLBW infants. At follow-up, cocaine-exposed VLBW children were delayed in cognitive, motor and language development compared to controls. Almost half (45%) of the exposed children scored in the range of mental retardation compared to 16% of the comparison VLBW children.

The persistent cognitive, motor and language delays of the cocaine-exposed VLBW children, combined with the poorer behavioral interactions of cocaine-using women with their infants in the neonatal period, indicate a need for increased developmental surveillance of cocaine-exposed VLBW infants with a focus on maternal drug treatment and parenting interventions. © 2001 Published by Elsevier Science Ireland Ltd.

Keywords: Cocaine; Very low birthweight; Parenting stress; Infant development; Language; Coping; Maternal–child interaction; Prematurity

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1. Introduction

Concerns about the long-term effects of prenatal cocaine and polydrug exposure on child developmental and health outcomes have precipitated innovative prevention and intervention programs in pediatric medicine and nursing [1,2]. Prenatal cocaine exposure is now recognized as a significant problem in both urban and rural neonatal nurseries in the United States, with national prevalence rates ranging from 10% to 20% [3,4]. Cocaine is known to cross the placental barrier antenatally and is thought to affect central nervous system development directly through its well-documented actions on major neurotransmitter systems and indirectly, through vasoconstriction and fetal hypoxemia [5,6].

Both animal and human studies have plausibly linked fetal cocaine exposure to later learning difficulties, although human outcome studies are still rare and controversial, particularly, beyond the first year of life, and the mechanisms of pathology are uncertain [7–9]. Fetal cocaine exposure has been linked to increased incidence of low and very low birthweight (VLBW), prematurity, intrauterine growth retardation, microcephaly, prenatal infections and subtle brain lesions neonatally, highlighting the need for enhanced efforts at health and developmental surveillance after birth [10–13]. A growing number of studies suggest that fetal cocaine exposure is related to later cognitive and motor impairments [14–19], but not all studies have found such deficits [20,21].

While longer-term developmental outcomes of cocaine-exposed cohorts are still unspecified, significant risk is implied in the known prevalence of polydrug use among cocaine users [11,12,14,22], the psychological disorders and impaired social interactions of maternal users [23–26] and the high levels of violence, child abuse and neglect characteristic of drug-using populations [27]. Thus, both biological and environmental correlates of maternal cocaine use may affect child developmental outcomes. Because fetal cocaine exposure is associated with prematurity and lower birthweight [11,28,29], cocaine-exposed infants may also be at increased risk for developmental problems known to be sequelae of prematurity, or very low birthweight and prematurity may pose special risk for the drug-exposed infant. Maternal interactions with her infant in the first year of life, which directly support child developmental competence may also be negatively affected by maternal substance abuse [30]. Because VLBW infants are at greater biological risk, drug-exposed VLBW infants may be at greater developmental risk.

Despite the potential for additional risk under conditions of prematurity, there are few studies that have addressed the relationship of fetal cocaine exposure to developmental outcomes in very low birthweight infants [28,29,31]. In two studies, in the neonatal period, cocaine-exposed infants differed from nonexposed, preterm infants by exhibiting more irritability and hormonal differences indicating greater stress, suggesting greater developmental risk [28,29]. Another earlier study [31] also found cocaine-exposed very low birthweight infants to have higher incidence of mild (Grades I–II) intraventricular hemorrhage, poorer cognitive and motor skills and a higher incidence of developmental delay at a mean corrected age of 17 months compared to similar race and SES nonexposed peers. However, standard infant developmental assessments are poor

predictors of later cognitive outcomes prior to 2 years of age. Moreover, the only prior follow-up utilized the old form of the Bayley Scales, with outdated norms [32].

Thus, the present study assessed a cohort of very low birthweight, cocaine-exposed infants and a comparison group of nonexposed infants who were identified at birth and followed to 3 years of age. Standard developmental outcome measures, early maternal–child interactions, as well as maternal psychological characteristics and environmental factors conceptualized to be important for child outcome were assessed.

2. Methods

All children and their biologic mothers or other caregivers were scheduled for follow-up as part of a longitudinal study of very low birthweight (VLBW) (<1500 g) infants. Admissions to the neonatal intensive care unit were prospectively reviewed for 2 years for consecutive recruitment of VLBW infants with bronchopulmonary dysplasia (BPD) and of VLBW infants without BPD who were of comparable race, social class, age and cocaine status. Cocaine status was determined through prospective urine screening or clinical interview at the time of the infant's birth, or both. Urine samples were obtained immediately before or after labor and delivery in the NICU in which the majority (85%) of infants were recruited. They were analyzed by enzyme immunoassay, using the Syva EMIT method (Syva, Palo Alto, CA), for the presence of cocaine's primary metabolite, benzoylecgonine and for heroin, phencyclidine, methadone, opiates, barbiturates and marijuana. The specificity for benzoylecgonine is 99% at concentrations of 0.3 mg/ml.

For this study, all VLBW infants with positive findings of maternal cocaine use were compared with an equal number of noncocaine-exposed VLBW infants of similar race, social class and age, from the same study population, all of whom were African–American and receiving public assistance. Groups were balanced for BPD status and did not differ in gender, socioeconomic status, or maternal age, but cocaine-using mothers were less likely to be married (Table 1). Two additional cocaine-exposed infants were identified, one, a white female, middle-class (Hollingshead class II) [33] survivor whose mother declined to participate in the study and, the second, a twin who died at birth and whose sibling entered the study. For 81% of the sample (33 cocaine-positive, 33 cocaine-negative), data from maternal self-report or urine screens were available on other drug use during pregnancy.

As is typical of other studies of drug-exposed infants, mothers who used cocaine were also more likely to report using cigarettes, alcohol and marijuana during pregnancy than nonusing mothers. A lower percentage of cocaine-exposed infants were in the care of their biologic mothers at follow-up. Groups did not differ in neonatal risk status or gender, socioeconomic status, or maternal age at birth, but cocaine-using mothers were less likely to be married. At neonatal enrollment, groups did not differ on an extensive number of perinatal risk variables, with the sole exception of incidence of Grades I–II intraventricular hemorrhage, which was higher in the exposed group.

This study was approved by the participating hospitals' Institutional Review Boards and maternal written informed consent was obtained for all subjects. Each family was given a 25-dollar (US\$25) stipend for completion of the visit.

Table 1
Sample characteristics at birth

	Cocaine-positive (n = 41)	Cocaine-negative (n = 41)	<i>t</i> / <i>x</i> ²	<i>p</i>
Race (% black)	41 (100%)	41 (100%)		
Sex (% male)	24 (58%)	19 (46%)	1.2	NS
Maternal marital status ^a	35 (85%)	27 (66%)	3.2	0.05
Social class ^b	4.5 ± 0.7	4.3 ± 0.7	0.9	NS
Maternal age (year) ^b	27.1 ± 4	25.6 ± 6	1.2	NS
Alcohol use ^c	15 (46%)	2 (6%)	13.3	0.001
Marijuana use ^c	12 (36%)	0	14.7	0.001
Opiate use ^c	1 (3%)	0	1.0	NS
Barbiturate use ^c	4 (12%)	3 (9%)	0.2	NS
Cigarette use ^c	26 (78%)	11 (33%)	13.6	0.001
Maternal PPVT-R score	79 ± 17	79 ± 13	0.1	NS
Placement				
With biologic mother	27 (63%)	39 (95%)	3.6	0.01
Died	3 (7%)	1 (2%)	0.9	NS
Relative placement	7 (16%)	0	2.7	0.01
Foster placement	4 (9%)	0	2.0	0.02
Adopted	2 (5%)	1 (2%)	0.5	NS

NS = Not significant.

^a Percentage unmarried.

^b Mean ± S.D.

^c Based on 81% of sample (33 cocaine positive and 33 negative).

2.1. Procedures

The following medical information was extracted from the NICU chart: infant gestational age (based on a combination of Ballard examination and dates from the last menstrual period) [34], birth weight (in grams), length and head circumference (cm), Apgar scores at 1 and 5 min and the presence or absence of respiratory distress syndrome and BPD (defined as requiring supplemental oxygen more than 28 days, with accompanying radiologic changes indicative of pulmonary disease). Also noted were the presence or absence of patent ductus arteriosus, necrotizing enterocolitis (proved, with or without surgery), retinopathy of prematurity and abnormal hearing test results; number of days ventilator support was required and number of days supplemental oxygen was used; peak bilirubin levels; and the presence or absence of seizures, periventricular leukomalacia and intraventricular hemorrhage. Echodense lesions were identified when a density was noted that was not an intraventricular hemorrhage. Ultrasound studies were obtained for all VLBW infants during their NICU hospitalization and were interpreted by one of four board certified pediatric radiologists, who were not informed of the infant drug status. For IVH, a rating of severity based on extent of lesion was devised: no hemorrhage on ultrasonography was scored as zero and lesions received gradings from one to four by the criteria of Papile et al. [35] All infants had at least one ultrasound study; ratings were based on the most severe lesion diagnosed if more than one study was done.

Attempts were made to enroll all cocaine-exposed VLBW infants and their non-cocaine-exposed VLBW comparison infants in the follow-up study. At the neonatal visit, the Nursing Child Assessment Feeding Scale (NCAFS) [36] was administered. All infants were videotaped as close to 40 weeks (gestational age) during feeding with their biological mothers. Videotapes were rated by one of two observers masked to infant drug status.

The NCAFS consists of 76 items rated dichotomously for the occurrence/nonoccurrence of specific observed parent and infant behaviors during a feeding interaction in the first year of life. The Parent Scale is composed of four subscales (sensitivity to cues, response to distress, socioemotional growth fostering and cognitive growth fostering) and the Infant Scale of two subscales (responsiveness to parent and clarity of cues). Higher scores are construed to reflect more optimal interactions. Validity and reliability are reportedly high, with Cronbach's alpha of 0.82 for the Infant total, 0.90 for the Parent total and individual subscales ranging from 0.70 to 0.88. Coders for the present study were trained to an interrater reliability of 0.80 by a certified NCAFS trainer.

The Peabody Picture Vocabulary Test (PPVT-R) [37], a brief screening measure used to assess maternal verbal comprehension, was also administered at the neonatal visit. Internal consistency ranges from 0.73 to 0.84 and test–retest reliabilities vary from 0.76 to 0.79. The PPVT-R is highly correlated with various IQ scales. Because drug-using women might have lower intellectual abilities and because maternal IQ relates highly to child outcomes, it was important to control for differences in this factor in evaluating child outcomes.

The COPE [38] was used to assess maternal-coping style and strategy. Subscales measure conceptually distinct aspects of problem focused coping. Cronbach's alphas for each scale are acceptably high (>0.6); test–retest reliabilities for subscales range from 0.42 to 0.89.

The following child measures were administered at 3 years.

The Bayley Scales of Infant Development [39] are widely used assessments of infant development. The Mental Development Index (MDI) is a standard score reflecting memory, learning and problem-solving abilities. The psychomotor index (PDI) measures gross and fine motor control and coordination. Because normative data on the Bayley scales yield a standard score range restricted from 50 to 140 (± 2 S.D.), lower scores were extrapolated [40].

The Battelle communication domain subscale [41] provides a standard measure of receptive and expressive language skills. The scales provide a deviation quotient similar to the standard scores of the Bayley scales.

2.2. Analyses

Groups were compared on medical risk variables, maternal characteristics and developmental outcomes with *t*-tests for continuous data, chi-square analyses for categorical variables, or *z*-tests, with correction for continuity, for proportions. For all child assessments, we hypothesized that cocaine-exposed children would have poorer behavioral ratings and developmental outcomes at follow-up, based on the outcome assessment at 17 months. Thus, these tests were one-tailed. All other tests were two-tailed. When cell sizes

were too small to use chi-square analysis, Fisher's exact test was used. Analyses of covariance were used to compare developmental outcomes with control for confounding variables, when necessary.

3. Results

Of 41 identified cocaine-exposed infants, three had died, the parents of six refused follow-up and one was visually impaired that could not be tested. Of the 41 nonexposed infants, one died, one was adopted and one was lost to the study. Thus, the attrition was higher in the cocaine-exposed cohort, with 82% of survivors seen from the exposed group and 95% of survivors seen from the nonexposed group. For maternal–infant interaction and maternal psychological measures, data from biologic mothers were included only.

3.1. Maternal–infant interactions

There was a nonsignificant trend for early parenting interactions of cocaine-using women, rated by observers masked to maternal drug use, to be less optimal, reflected in the significantly lower levels of socioemotional growth fostering of the cocaine-using mothers (see Table 2) during feeding. There were no differences in ratings of maternal sensitivity to infant cues or in maternal responses to infant distress. There was a nonsignificant trend for cocaine-exposed infants to be rated as less responsive during interactions.

3.2. Maternal characteristics

There were no differences by group in maternal vocabulary scores (see Table 1). As noted in Table 3, cocaine-using mothers who retained custody of their VLBW infants exerted greater overall coping efforts than noncocaine-using mothers. These coping efforts

Table 2
Maternal infant interactions for cocaine-exposed and noncocaine-exposed infants at neonatal visit on NCAFS

NCAFS subscale	Cocaine-positive (<i>n</i> = 19)	Cocaine-negative (<i>n</i> = 39)	<i>t</i>	<i>p</i> ^a
	Mean ± S.D.	Mean ± S.D.		
Sensitivity to cues	11.5 ± 2	12.1 ± 2	− 1.1	NS
Response to distress	9.2 ± 2	9.5 ± 2	− 0.5	NS
Socioemotional growth fostering	10.2 ± 2	11.4 ± 2	− 2.1	0.025
Cognitive growth fostering	6.8 ± 1	6.2 ± 2	1.4	NS
Total parent	37.6 ± 6	40.0 ± 6	− 1.3	0.10
Clarity	11.3 ± 3	11.1 ± 2	0.3	NS
Responsivity	5.8 ± 2	6.7 ± 2	− 1.3	0.10
Total child	17.1 ± 5	17.7 ± 4	− 0.6	NS

NS = Not significant.

^a One-tailed.

Table 3
Differences in maternal coping mechanisms (COPE) between cocaine-using and -nonusing mothers

COPE subscale	Cocaine-positive (<i>n</i> =17)	Cocaine-negative (<i>n</i> =37)	<i>t</i>	<i>p</i>
	Mean ± S.D.	Mean ± S.D.		
Active coping	12.3 ± 2	10.9 ± 3	2.1	0.05
Planning	12.9 ± 3	11.9 ± 3	1.2	NS
Suppression	11.0 ± 3	9.2 ± 2	2.3	0.05
Restraint	11.4 ± 2	9.2 ± 2	3.2	0.005
Instrumental social support	13.0 ± 3	10.2 ± 3	3.2	0.005
Emotional social support	10.9 ± 2	10.6 ± 3	0.4	NS
Positive reinterpretation	12.9 ± 3	12.1 ± 3	1.1	NS
Acceptance	11.6 ± 3	10.8 ± 3	0.9	NS
Religion	14.2 ± 2	12.2 ± 3	2.3	0.05
Focus on venting	11.5 ± 3	10.0 ± 4	1.4	NS
Denial	7.5 ± 3	5.8 ± 2	2.3	0.05
Behavioral disengagement	6.9 ± 3	5.5 ± 2	2.3	0.05
Mental disengagement	7.9 ± 3	7.5 ± 3	0.5	0.7
Alcohol/drug	5.8 ± 2	4.8 ± 2	1.7	0.09
Humor	7.8 ± 3	6.1 ± 2	2.3	0.03
Total	157.5 ± 25	136.7 ± 20	3.3	0.002

NS = Not significant.

included some behaviors considered to be maladaptive, such as denial, behavioral disengagement, alcohol and drug use. However, cocaine-using mothers also reported greater reliance on religion, active coping, humor, utilization of instrumental social supports, restraint and suppression of attention to other activities, all conceptualized to be adaptive efforts [38].

3.3. Child mental, motor and language outcomes

To assess potential effects of attrition, we subjected all medical and demographic variables documented at birth to a series of *t*-tests to determine whether the groups differed at the follow-up at 3 years in terms of birth and demographic characteristics. The cocaine-exposed group was more likely to have a history of intraventricular hemorrhage based on ultrasound findings in the neonatal period, consistent with the overall sample (see Table 4). All other medical variables were not different. At follow-up, one child in the nonexposed group had a diagnosis of cerebral palsy. Children tested at follow-up did not differ from those not tested on any variable.

Children who had been cocaine-exposed performed more poorly in both cognitive and motor skills when mean scores were compared (see Table 5). Because controlling for the effects of IVH did not reduce the effects of cocaine exposure on MDI or PDI, unadjusted data are presented. Mean MDI scores were in the risk range for the drug-exposed group, while in the low normal range for the nonexposed group. Motor scores were in the low normal range for the drug-exposed group, while in the average range for the nonexposed group. A higher percentage of cocaine-exposed VLBW children than nonexposed VLBW children had scores in the range of mental and motor retardation (<70 standard score).

Table 4
Neonatal complications for follow-up group

	Cocaine-positive (<i>n</i> = 31)	Cocaine-negative (<i>n</i> = 38)	<i>t/x</i> ²	<i>p</i>
Birthweight	1026 ± 233	998 ± 244	0.50	NS
Gestational age	28 ± 2	28 ± 2	1.2	NS
Apgar (1 min)	4 ± 2	4 ± 3	0.01	NS
Apgar (5 min)	6 ± 2	6 ± 2	0.04	NS
Duration of ventilator support	20 ± 18	21 ± 22	0.15	NS
Respiratory distress	26 (84%)	31 (82%)	0.06	NS
Apnea	27 (87%)	30 (79%)	0.79	NS
Intraventricular hemorrhage	17 (55%)	12 (32%)	3.8	0.05
Echodense lesions	10 (32%)	6 (16%)	2.6	NS
Periventricular leukomalacia	2 (7%)	2 (5%)	0.07	NS
Hydrocephalus	6 (19%)	4 (11%)	1.1	NS
Seizures	1 (3%)	1 (3%)	0.02	NS
Patent ductus arteriosus	12 (39%)	16 (42%)	0.08	NS
Retinopathy of prematurity	9 (29%)	11 (29%)	0.00	NS
Necrotizing enterocolitis	4 (13)	2 (5%)	1.3	NS
Tracheotomy	0	1 (3%)		NS
Home oxygen	0	1 (3%)		NS

NS = Not significant.

The rate of mental retardation was increased fivefold in the exposed group and the rate of motor retardation threefold.

The association of cocaine exposure and MDI and PDI outcomes were also quantified using relative risk ratios with four sets of assumptions, i.e. (1) that all infants with

Table 5
Developmental outcomes of cocaine-positive and cocaine-negative VLBW children

	Cocaine-positive (<i>n</i> = 31)	Cocaine-negative (<i>n</i> = 38)	<i>t/x</i> ²	<i>p</i>
	Mean ± S.D.	Mean ± S.D.		
Corrected age (months)	35 ± 7	35 ± 6	0.4	NS
Bayley MDI	75 ± 24	85 ± 17	− 1.8	0.05
MDI < 70	14 (45%)	6 (16%)	5.4	0.01
PDI	83 ± 25	96 ± 16	− 2.3	0.01
PDI < 70	9 (29%)	2 (5%)	5.3	0.01
Battelle language scale	(<i>n</i> = 25)	(<i>n</i> = 30)		
	Mean ± S.D.	Mean ± S.D.		
Expressive quotient	78 ± 11	91 ± 3	− 3.2	0.001
Receptive quotient	78 ± 13	81 ± 14	− 0.6	NS
Total communication	75 ± 12	84 ± 17	− 2.7	0.005

MDI = Mental Development Index

PDI = Psychomotor Development Index

NS = Not significant.

missing data had good outcomes (standard score >70); (2) that all infants with missing data had outcomes <70; (3) that missing control infants had scores <70 and that cocaine-exposed infants had scores ≥ 70 ; and (4) that missing data are dropped from analyses. Under these conditions, the relative risk ratios for MDI are 2.46 (95% CI: 1.05, 5.73), 2.76 (95% CI: 1.40, 5.47), 1.84 (95% CI: 0.87, 3.89) and 2.86 (95% CI: 1.25, 6.56). For PDI, comparable relative risk ratios are 4.74 (95% CI: 1.09, 20.53), 4.21 (95% CI: 1.55, 11.46), 2.37 (95% CI: 0.80, 7.05) and 5.52 (95% CI: 1.29, 23.68).

Cocaine-exposed children also performed more poorly on overall communication skills development, although both groups of VLBW children performed in the risk range. The low overall scores of the cocaine-exposed group were due to relatively greater deficits in expressive language skills, which were in the risk range for cocaine-exposed VLBW children, but within normal limits for the nonexposed group. Receptive language skills were not different between the groups.

The differences in cognition, motor and language scores of the cocaine-exposed group were not diminished when data analyses were rerun to control for the effects of IVH, the only neonatal neurologic complication which differed between the groups. Thus, when the baseline differences in this complication were controlled, the effects of cocaine on these developmental outcomes remained significant.

4. Discussion

Three-year-old children with a history of very low birthweight and cocaine exposure were delayed in cognitive, motor and language development relative to nonexposed VLBW children of similar age, race and socioeconomic status. Almost half of the exposed children scored in the range of mental retardation, nearly three times the rate of the nonexposed VLBW children who were matched for ethnicity and social class. In the neonatal period, cocaine-exposed VLBW infants tended to be rated as less responsive in interactions with their biological mothers and their mothers were rated as less nurturing and emotionally available. Their mothers were also more likely to exert greater coping efforts overall and to use some coping mechanisms considered less adaptive and associated with less optimal parenting practices [42].

These findings are consistent with other studies of preterm and term cocaine-exposed children, which have been demonstrated with cognitive, language, motor and behavioral deficits compared to nonexposed children [15–17,28,31,41,43]. Rates of mental and motor retardation were significantly higher in this sample of cocaine-exposed VLBW infants compared to nonexposed VLBW infants, who were already at risk for developmental disabilities due to complications of prematurity [44]. Moreover, these findings document persistent deficits to 3 years of age, a time when assessments of intelligence are predictive to school age.

In the only other study of cocaine-exposed VLBW infants [31], neurologic, behavioral and hormonal differences were noted in the neonatal period in the cocaine-exposed group in comparison to nonexposed infants. In that study, cocaine-using mothers touched and held their infants less frequently than mothers of nonexposed infants, consistent with the present findings of lower maternal social and emotional growth fostering behaviors during

feeding. In particular, this finding contrasts with studies of nondrug-using mothers of preterm infants in the neonatal period, which document increased maternal activity and interactive behaviors [45]. In another study, preterm infants with cocaine exposure were also found to be more irritable than their nonexposed counterparts, perhaps contributing to the trend towards poorer responsiveness in their interactions demonstrated in the present study [29]. The lower responsiveness of cocaine-exposed VLBW infants in this study fits well with the hypothesis that fetal cocaine exposure may have specific effects on arousal regulation mechanisms in infants, which have implications for learning and memory [5,8,44,46,47].

One limitation of the present study is that cocaine-exposure was documented only through maternal clinical interviews and urine screens were not available for all infants. Thus, some infants in the comparison group could have been exposed without detection. However, such misidentification would have only served to decrease the detection of differences between exposure groups. Another limitation is that, as in other studies, cocaine users were more likely to use other drugs also, especially tobacco, alcohol and marijuana, than the nonusing mothers. Because exposure to these drugs was not reliably quantified, negative sequelae found in this study could be due to effects of these other drugs or to their interactive effects with cocaine, rather than to cocaine exposure per se. Alcohol, tobacco and marijuana have all been noted to have independent negative effects on various domains of child developmental outcome [48,49].

Finally, for some maternal measures, because cocaine-exposed children were more likely to be placed outside biological maternal care, the loss in maternal sample size may have reduced the power of the study to detect subtle differences in maternal behaviors and attitudes.

Because more cocaine-using women had lost custody of their infants, results from this study related to maternal–infant interactions and maternal-coping mechanisms may not be generalized to mothers who are heavier cocaine users or to users who have lost custody of their infants. Because of the differences between cocaine-positive and -negative children in maternal custody, it is also not possible to infer whether maternal–infant interactions and coping mechanisms contributed to the poorer outcomes of the drug-exposed group. However, in other populations, maternal interactions with her child have been demonstrably related to child developmental competence [50].

Nevertheless, findings from this study document persistent cognitive, motor and language delays in cocaine-exposed VLBW children at 3 years and poorer behavioral interactions of cocaine-using women with their infants in the neonatal period, warranting increased developmental surveillance of cocaine-exposed VLBW infants with a focus on maternal drug treatment and parenting interventions.

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