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Longitudinal Study of Maternal Report of Sleep Problems in Children with Prenatal Exposure to Cocaine and Other Drugs

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Abstract

Sleep data were collected by maternal report in a prospective longitudinal follow-up of cocaine exposed and unexposed children. There were 139 subjects: 23 with no prenatal drug exposure, 55 exposed to cocaine alone or in combination with other drugs, and 61 exposed to drugs other than cocaine. Characteristics differed between exposure groups, including birth size, caretaker changes, and maternal SES and postnatal drug use. Compared to those with no drug exposure, children with prenatal drug exposure other than cocaine experienced greater sleep problems (mean [SD], 5 [4.93] vs 7.7 [4.85], $p = .026$). Prenatal nicotine exposure was a unique predictor of sleep problems ($R^2 = .028$, $p = .048$). Early sleep problems predicted later sleep problems (all p 's $< .01$). Together, these preliminary findings suggest possible neurotoxic sleep effects that persist over time. Larger studies, however, need to be conducted that better control for potential postnatal confounding factors.

Introduction

The 2005–2006 National Survey on Drug Use and Health reported that 4% of pregnant women between the ages of 15 and 44 admitted to illicit drug use, 2.9% to binge drinking, and 16.5% to tobacco use (“Substance Abuse and Mental Health Services Administration, 2007,”). For nonpregnant women of the same age range the percentages are 10, 23.6, and 29.5, respectively. The last decade has shown a convergence of moderate and specific biopsychosocial effects due to prenatal cocaine exposure (PCE; Glantz & Chambers, 2006), but little is known about the potential role of sleep in the development of children with PCE and other prenatal drug exposure. Sleep, the predominant behavior state of neonates (Mirmiran, Maas, & Ariagno, 2003), is a measurable neurobehavioral state with the potential to inform our understanding of specific drug effects on development. Studies of sleep problems in infants with PCE have shown less sleep (DiPietro, Suess, Wheeler, Smouse, & Newlin, 1995; Gingras, Feibel, Dalley, Muelenaer, & Knight, 1995; Regalado, Schechtman, Del Angel, & Bean, 1995, 1996), shorter sleep bouts (DiPietro et al., 1995), more arousals during sleep (Gingras et al., 1995), more time in transitional/drowsy state (DiPietro et al., 1995), and more intermediate sleep (Regalado et al., 1995). Increased wake time and awakenings during the night were found in 3-years-olds with prenatal marijuana exposure (Dahl, Scher, Williamson, Robles, & Day, 1995), and global sleeping problems and differences in EEG patterns have been found in infants with prenatal exposure to opiates (Hutchings, 1982), alcohol (Chiriboga, 1993), and nicotine (Pichini & Garcia-Algar, 2006).

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Preclinical studies as well as theoretical models of the neuropharmacodynamics of cocaine suggest that PCE affects monoaminergic pathways involved in sleep/wake regulation (Mayes, 1999). Infants with PCE have difficulty regulating behavior states (e.g., they spend more time in indeterminate sleep than in active sleep), suggesting that cocaine may delay central nervous system maturation (Gingras et al., 1995). Furthermore, it has been suggested that the effects of prenatal drug exposure may wax and wane throughout life (Dow-Edwards, Mayes, Spear, & Hurd, 1999; Stone et al., 1996). For example, sleep decrements associated with prenatal alcohol exposure may manifest during periods of vulnerability, such as infancy and adolescence (Stone et al., 1996).

There are no studies of the effects of prenatal exposure to cocaine, opiates, alcohol, or nicotine on sleep beyond the first year of life. The only longitudinal study of prenatal marijuana exposure found worse sleep in the exposed than in the control group at age 3 as measured by polysomnography (Dahl et al., 1995). In other populations, poor sleep from infancy to adolescence has been correlated with negative short- and long-term outcomes in areas such as sleepiness (Carskadon, 1982; Carskadon et al., 1980), activity and social interest (Carskadon et al., 1980), psychomotor performance (Anders, Keener, & Kraemer, 1985), behavioral disturbance (Thoman, Denenberg, Sievel, Zeidner, & Becker, 1981), memory and learning (Carskadon, Acebo, & Jenni, 2004), an substance use (Carskadon, 1990). Furthermore, using sleep and health questionnaires and the Child Behavior Checklist (CBCL), a large study of children 4–12 years of age found that the best predictor of current sleep problems was a history of sleep problems before age 2 (Stein, Mendelsohn, Obermeyer, Amromin, & Benca, 2001). The longitudinal study of sleep in children with prenatal cocaine and other drug exposure could help clarify whether or not there are neurotoxic neonatal sleep effects that persist over time.

Prenatal exposure to illicit drugs has been hard to extricate from correlates of drug-abusing mothers, such as other drug use during pregnancy (Glantz & Chambers, 2006). This difficulty dictates empirical regard for the amalgamation of pre- and postnatal environmental exposures and inherent vulnerabilities when investigating risk in these children. Race and socioeconomic status (SES) are the most common correlates of sleep disturbance (i. e., being African American and being of a lower SES predict more childhood sleep disturbance) emerging from the few studies investigating the demographic, socioeconomic, and environmental determinants of childhood sleep problems (Buckhalt, El-Sheikh, & Keller, 2007; El-Sheikh, Buckhalt, Keller, Cummings, & Acebo, 2007; McLaughlin Crabtree et al., 2005). These factors are also correlated with specific illicit drug use during pregnancy and are, thus, important to consider when investigating risks of children with prenatal drug exposure. The specific nature of these risks remains obscure and could be clarified by broadening our attention to the area of sleep.

Our study follows the sleep of children with prenatal exposure to cocaine and other drugs into late childhood. We studied sleep problems, as measured by maternal report, from 18 months to 9 years of age in children with prenatal exposure to cocaine with or without prenatal exposure to other drugs (i. e., opiates, marijuana, alcohol, or nicotine); children with prenatal exposure to drugs other than cocaine (i. e., opiates, marijuana, alcohol, or nicotine); and children with no prenatal drug exposure. We also studied the relationship between early sleep problems and later sleep problems in these children.

Methods

Study Design

This study is based on data from the Maternal Lifestyle Study (MLS), a longitudinal, multi-site investigation of the effects of prenatal cocaine exposure on child developmental outcome. (Bada et al., 2002; Bauer et al., 2005; Lester et al., 2001). The largest prospective study of this kind, the MLS enrolled 1388 children (658 in the cocaine/opiate-exposed group and 730 in the

comparison group) at birth in Detroit, MI, Memphis, TN, Miami, FL and Providence, RI, in 1993–1995 (Lester et al., 2002). The MLS was approved by an appropriate Institutional Review Board at each site. A NIDA Certificate of Confidentiality was obtained that assured confidentiality of information regarding the participants' drug use. Mothers were initially recruited in the hospital after delivery and informed consent was obtained at that time.

Participants were recruited to maximize likelihood of having prenatal cocaine exposure or to be a group matched control. Therefore, all women delivering very low birth weight infants of 501 to 1500 g were identified and recruited whereas mothers delivering low birth weight and normal birth weight infants were recruited during normal hours of operation (i.e., excluding nights and weekends). Mothers were screened for eligibility based on the following criteria: 18 years or older and without psychiatric disorders, developmental delays, or language barriers. Neonates were eligible provided that they were inborn, likely to survive, singleton, and < 43 weeks gestational age. Mother-infant dyads were also excluded from the longitudinal study, which started with the 1-month visit, if the infant has a chromosomal abnormality or TORCH (toxoplasmosis, rubella, cytomegalovirus, herpes, and syphilis) infection or when the mother planned to move out of the catchment area. Comparison participants were group matched on ethnicity (black, white, Hispanic, other), gender, and gestational age (within 2 weeks). When an infant in the comparison group did not attend the 1-month visit, another match was generated until a comparison infant was successfully enrolled or the recruitment period ended. It was not possible, however, to recruit and enroll replacement exposed participants if an exposed infant withdrew, which accounts for the uneven groups. The MLS conducted 9 follow-up visits from 18 months to 9 years. Because some of the sleep measures were only administered in the Providence sample, this study involves data from the Providence cohort only.

Participants

The Providence cohort of the MLS ($n = 208$, 106 in the cocaine/opiate-exposed group and 102 in the comparison group) was recruited from Women & Infants' Hospital. Mothers were assigned to the cocaine/opiate exposure group based on maternal report and/or meconium toxicology with gas chromatography-mass spectroscopy assay confirmation. Infants were assigned to the comparison group when mothers denied cocaine/opiate use during pregnancy and the meconium toxicology was negative. The same procedure applied for determination of marijuana exposure. Alcohol and tobacco use were determined by maternal report alone.

Of the original 208 participants, 162 were administered the Providence-only sleep measures. Of these, 139 (86%) were seen at age 9. For this study, in order to compare children with cocaine exposure to children with other drug exposure, the 139 children were divided into three groups: the prenatal cocaine exposure (PCE) group of children with PCE alone or combined with other drugs ($n = 55$; i. e., opiates, marijuana, alcohol, or nicotine); the other drug (OD) group of children exposed to drugs other than cocaine ($n = 61$; i. e., opiates, marijuana, alcohol, or nicotine); and the unexposed (UN) group of children with no prenatal drug exposure ($n = 23$).

Demographic and Maternal Measures

A questionnaire administered to the mothers at the 1-month visit was used to collect information about prenatal care, maternal age, marital status, education level, SES, and poverty status. Postnatal use of substances was assessed using the Caretaker Inventory of Substance Use (CISU) at yearly visits, from which we computed dichotomous variables of whether or not the mother/caretaker used each of the 5 substances since pregnancy each year through year 9. Questionnaires were also used to measure number of caretaker changes (number of times the child changed caregivers over the 9-year period), child abuse (yes/no, each year, defined by medical exam findings suggestive of physical/sexual abuse or removal of the child from the

home due to suspicion or verification of physical/sexual abuse) and domestic violence including physical and sexual abuse experienced by the caretaker (yes/no, years 3–9). Maternal psychological distress was measured with the Brief Symptom Inventory (Derogatis, 1993), a 53-item shortened version of the Revised 90-item Symptom Checklist in which higher scores reflect more psychological distress. The BSI was administered at 4 months, 30 months, 5 years, and 9 years. Maternal depression was assessed using the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), a 21-item self-report scale that measures symptoms of depression.

Sleep Measures

The Family Interest Interview (FII) was administered in Providence only, at 18 and 30 months. From the FII the following 10 items were scored as 0 for “no” and 1 for “yes” across varying time frames from “usually” to “during the past week:” having problems when falling asleep at night, having a bedtime after 9 pm, fighting about going to bed for longer than 30 minutes, having no regular bedtime routine, falling asleep alone in own bed < 4 nights a week, sleeping with an adult for most of the night, sleeping with an adult being identified as a problem, waking up > 2 times nightly, waking/calling out 3 or more nights a week, and not getting enough sleep. The toddler was assigned 1 point if each of the sleep problems occurred at either age. The CBCL was administered to the mother when the child was 3, 5, 7, and 9 years old. The CBCL is a standardized questionnaire used to identify behavioral problems of children over the past 6 months (Achenbach, 1991) and includes the following sleep-related items: having problems sleeping at night, having trouble getting to sleep, talking in sleep, crying in sleep, sleepwalking, waking up often at night, and sleeping less than or more than most. The child was assigned 1 point for each item endorsed by the parent. The general health questionnaire contained the following sleep items: problems with sleeping at night, falling asleep at a reasonable time, and waking at night requiring attention. The child was assigned 1 point for each item endorsed by the parent. All items within 3 time periods (i.e., toddler [18–30 months], preschool [ages 3–5], and school-aged [ages 6–9]) were summed for 3 age-relative indices of sleep problems, each ranging 0–10. We then computed a total sleep problem index summing all 30 items from 18 months to 9 years. This specific procedure of creating a composite score from the CBCL and other questionnaires has been used in other studies (e. g., Alfano, Ginsburg, & Kingery, 2007; Storch et al., 2008); adequate internal consistency has been demonstrated ($\alpha = .64$; Alfano et al., 2007) using this method. The Cronbach’s alphas for the sleep problem index at each time period – toddler, preschool, and school-aged – for our study were as follows: .67, .74, and .79 respectively. The Cronbach’s alpha for the overall sleep problem index was .835.

Statistical Analyses

Chi-square and *t*-tests were used to compare sample characteristics related to attrition (Table 1) and exposure groups (Table 2). Analysis of variance (ANOVA), followed by post-hoc *t*-tests, was used to compare the three groups on the overall sleep problem index. Linear regression was used to determine unique variance of each drug. Covariates were selected from the variables in Tables 1 and 2 if they (a) differed significantly between exposure groups, (b) were associated with a given sleep outcome at $p \leq .2$, and (c) were not highly correlated with other covariates (i.e., $r > .7$). Only the number of caretaker changes measure met all three criteria. Variables potentially on the causal pathway from PCE to sleep problems (i.e., gestational age, birth weight, head circumference, and length at birth) were not considered as covariates (Tu, West, Ellison, & Gilthorpe, 2005). Pearson correlations were used to investigate the association between early and later sleep problems.

Results

There were no significant differences (Table 1) on infant characteristics between subjects not seen at the 9-year visit ($n = 23$) and subjects seen at the 9-year visit ($n = 139$). Mothers or caregivers of participants no longer enrolled by year 9 were less likely to be in the lowest SES category and less likely to drink alcohol, smoke cigarettes, or report domestic violence (Table 1). Mothers reporting drinking more than 1 drink a day ranged from 2.3% at 2 years to 12.1% at 9 years.

There were differences among the three exposure groups (Table 2). Compared with the OD group, the PCE group was more likely to also have prenatal marijuana and nicotine exposure and less likely to have medical prenatal care; weighed less with smaller head circumference at birth; and had more caretaker changes during the 9-year period. The PCE group was also more likely to have unmarried mothers with income below the poverty line who had less than a high school education and reported more nicotine use and less alcohol use over the 9-year period. Compared to the UN group, the PCE group was less likely to have medical prenatal care and married mothers; was more likely to have mothers that used nicotine during the 9-year period; and had more caretaker changes during the 9-year period. Finally, compared to the UN group, the OD group was less likely to have mothers with income below the poverty line and with low SES and more likely to have mothers that used alcohol, nicotine, and opiates during the 9-year period. There were no significant differences in clinical level of psychiatric symptoms among exposure groups at any time point. The rate of mothers experiencing clinical levels of psychiatric symptoms across the 9 years of study ranged from 28% to 43%. The percentage of mothers expressing depression symptoms at a severe level at any time point across the 9 years did not differ by exposure group. Twenty-three percent of the sample expressed a severe level of depression during at least one assessment.

Differences on the overall sleep problem index (i.e., 18 months–9 years), were found among the three groups with number of caretaker changes entered as a covariate ($F [2, 139] = 4.522$, $p = .013$). There were more maternal reports of sleep problems in the OD group than in the UN group (mean [SD], 7.7 [4.85], 5 [4.93], respectively; $t [82] = 2.27$, $p = .026$). There were no differences in sleep problems between the two exposure groups or between the PCE group (M [SD], 6.63 [4.82]) and the UN group.

To determine the effects of each drug, we regressed all 5 prenatal drug exposure variables (presence or absence of cocaine, opiate, marijuana, alcohol, and tobacco) on the overall sleep problem index in a stepwise fashion. Exposure to nicotine during pregnancy was the only substance related to more sleep problems on the overall sleep problem index ($R^2 = .028$, $\beta = 1.65$, $p = .048$). To determine if this effect could be due to passive exposure due to maternal postnatal cigarette use, the regression analysis was repeated with maternal postnatal smoking (dichotomized as any/no smoking over the 9-year period) in the model. Prenatal smoking effect was marginally significant ($R^2 = .028$, $\beta = 1.78$, $p = .055$). However, postnatal smoking only explained .1% of the variance.

Finally, we tested whether early sleep problems predicted later sleep problems. Pearson correlations revealed that more sleep problems at each time period (i.e., toddler, preschool, school-age) were related to more sleep problems at every other time period (Table 3).

Comment

We found more sleep problems from 18 months to 9 years in children with prenatal drug exposure than in children with no prenatal drug exposure with adjustment for covariates. The addition of prenatal cocaine exposure to prenatal exposure to other drugs does not appear to increase sleep problems. Other studies have found effects of cocaine on sleep (DiPietro et al.,

1995; Gingras et al., 1995; Legido, Clancy, Spitzer, & Finnegan, 1992; Regalado et al., 1995, 1996). However, there are several differences between our study and previous studies that could explain discrepant findings. First, some studies (DiPietro et al., 1995; Gingras et al., 1995; Regalado et al., 1996; Scher, Richardson, & Day, 2000) did not control for other prenatal drug exposure. Second, other studies were restricted to infancy whereas we studied sleep problems from 18 months to 9 years. Third, our study was based on maternal report whereas other studies used objective measures of sleep including behavioral observation of sleep states and EEG measures of sleep architecture (DiPietro et al., 1995; Legido et al., 1992; Regalado et al., 1995, 1996). It would seem useful to include both subjective and objective measures in future studies of the effects of PCE on sleep.

There does appear to be a unique relationship between prenatal nicotine exposure and sleep problems that is not explained by maternal postnatal cigarette smoke exposure. There have been fewer studies of the effects of prenatal nicotine exposure on sleep than other drugs, such as cocaine and alcohol. Findings from animal studies of prenatal nicotine exposure include abnormal respiration during sleep (Hafstrom, Milerad, & Sundell, 2002) as well as abnormal maturation of sleep (Frank, Srere, Ledezma, O'Hara, & Heller, 2001). A recent study found that infants drinking breast milk containing nicotine slept significantly less afterwards than when sleeping after drinking breast milk without nicotine (53 vs. 85 minutes, $p < .001$; Mennella, Yourshaw, & Morgan, 2007). Furthermore, greater nicotine doses in the milk lead to less active sleep ($r = -0.60$, $df = 12$; $p = .02$). Smoking is associated with sleep disturbance in both adolescents (Patten, Choi, Gillin, & Pierce, 2000) and adults (Zhang, Samet, Caffo, & Punjabi, 2006). Nicotine may affect sleep directly by suppressing pontogeniculooccipital spike activity, a factor important in initiating and maintaining active sleep (Vazquez, Guzman-Marin, Salin-Pascual, & Drucker-Colin, 1996) or indirectly by inhibiting sleep-promoting neurons in the ventrolateral preoptic area. (Saint-Mleux et al., 2004).

A better understanding of the role of prenatal nicotine exposure alone and of its interaction with other drugs would help clarify our findings. That prenatal nicotine exposure is more damaging to sleep compared to other prenatal drug exposure cannot be concluded from the current study. Possibly, the level of prenatal exposure to nicotine was higher than cocaine or other substances. Future studies analyzing level of exposure would enable more confident comparisons across drug type. Furthermore, future studies should measure level of postnatal cigarette exposure (including others in the home) in order to more fully investigate our preliminary finding that prenatal nicotine exposure affects sleep independent of postnatal nicotine exposure.

Finally, we found a strong association between early and later sleep problems, which is consistent with studies in other populations (Zuckerman, Stevenson, & Bailey, 1987). The fact that sleep problems persist from early to later childhood with and without prenatal drug exposure is important. Poor sleep can cause, perpetuate, and/or exacerbate daytime sleepiness and cognitive problems as well as a wide array of psychological and physical health problems (Colten, 2006). Together, our findings suggest neurotoxic sleep effects that persist over time. Prenatal drug exposure accounted for a small but significant percent of the variance – a reminder that other factors, such as the environment, also play a role in brain development and sleep. Furthermore, it is possible that sleep problems mediate some of the effects of prenatal drug exposure on cognitive functioning and behavior problems (LaGasse et al., 2004). If so, proactive attention to sleep hygiene and screening for and treatment of sleep problems could reduce some of the adverse consequences of prenatal drug exposure. For example, adolescent substance use has been linked to both prenatal drug exposure (Glantz & Chambers, 2006) and poor sleep (Carskadon et al., 2004). Could effective sleep treatment reduce drug use among these at-risk youth?

Our study has several limitations. The sample size may not have been large enough for a study of this complexity and limited our ability to detect small effects. However, multicollinearity did not limit our regression analyses: the correlations among the 5 exposure variables and the correlations among the pre- and postnatal drug use variables were all $< .6$. It is also possible that amount rather than presence or absence of prenatal drug exposure would show different results. These data were available on too few participants, however, for appropriate statistical analysis. A larger sample size not only would have made it possible to investigate level of exposure but also would have made it possible to enter additional potential confounds as covariates if they were significantly different between groups without having to rely on other empirical criteria to maintain adequate power. Nevertheless, we used accepted theoretical and empirical criteria to evaluate a number of potential confounds. Sleep effects were found above and beyond number of caretaker changes – results which merit further investigation of this important topic. As mentioned above, our sleep measures were based on maternal report and could have been augmented with objective sleep measures. Regarding mothers with a history of substance misuse, little is known about the validity of subjective reporting of children's behaviors. Studies have found, however, that postnatal self-report measures of maternal cocaine use is as effective as antenatal measures in predicting neurobehavioral outcome (Jacobson & Jacobson, 1990). Finally, whereas we had biochemical verification of prenatal substance use, we relied on self-report of postnatal substance use.

Despite these limitations, this is the first longitudinal investigation into late childhood of the effects of prenatal cocaine and other drug exposure on sleep. The study of prenatal drug exposure on sleep offers insights into mechanisms of drug effects, may explain mediating effects of substances on other outcomes, and has treatment implications. We know that sleep disorders are amenable to treatment and that such treatments lead to positive mental and physical health outcomes. Gaining a better understanding of risk for sleep problems in children with prenatal drug exposure will help us equip them with the protective factor of good sleep.

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Table 1

Sample Characteristics by Attrition

	With 9yr data * n = 139	Without 9yr data * n = 23	p
Maternal Characteristics			
Race ¹			.374
White	68 (48.9%)	16 (69.6%)	
Black	38 (27.3%)	2 (8.7%)	
Hispanic	26 (18.7%)	4 (17.4%)	
Other	7 (5.1%)	1 (4.3%)	
Low SES (Hollinghead 5), 1m	23 (17.3%)	0 (0%)	.035
Below poverty line, 1 m	51 (40.2%)	7 (31.8%)	.459
Married	44 (31.9%)	13 (59.1%)	.076
Education < high school	44 (31.7%)	5 (21.7%)	.325
No prenatal care	9 (6.5%)	0 (0%)	.209
Age	29.1 (5.5)	29.1 (5.1)	.989
Prenatal drug use (yes)			
Cocaine	55 (39.6%)	6 (26.1%)	.216
Opiates	19 (13.75%)	6 (26.1%)	.127
Marijuana	20 (14.4%)	3 (13%)	.864
Alcohol	82 (59%)	9 (39.1%)	.075
Nicotine	74(53.2%)	9 (39.1%)	.21
Postnatal Environment			
Postnatal drug use (yes)			
Cocaine ²	0 (0%)	0 (0%)	-
Opiates	2 (1.4%)	1 (4.3%)	.338
Marijuana	5 (3.6%)	0 (0%)	.356
Alcohol	75 (54%)	2 (8.7%)	< .001
Nicotine	45 (32.4%)	2 (8.7%)	.02
Domestic Violence, 3–9yr	21 (15.1%)	0 (0%)	.046
Child abuse, 18m–9yr,	15 (10.8%)	1 (4.3%)	.337
# of caretaker Δ, 18m–9yr	0.58 (1.12)	0.43 (0.59)	.538
Newborn Medical Characteristics			
Premature (yes)	45 (32.4%)	9 (39.1%)	.524
Birth weight, g	2854.1 (805.5)	2856.7 (734.4)	.988
Length, cm	48.1 (4.94)	47.5 (3.8)	.602
Head circumference, cm	33 (2.7)	33.1 (2.4)	.947
Male	75 (54%)	14 (61%)	.537

* Mean (SD) or n (%)

¹ Race was identified by mother.

² Because mothers in the Providence cohort using cocaine during pregnancy were required by Rhode Island state law to remain abstinent from cocaine in order to be reunified with their children, it is feasible that no mother/caregiver in our sample used cocaine postnatally. Either the mother remained abstinent from cocaine and was able to be reunified with the child or the child was raised by a caregiver that reported abstinence from cocaine. Admitting to cocaine use postnatally to our research team should not have been more threatening than admission of prenatal cocaine use. A National Institute on Drug Abuse Certificate of Confidentiality ensured confidentiality of information regarding the participant's drug use. The certificate superseded the mandatory reporting of illegal substance use that was in effect in Rhode Island and was explained in full to the mothers.

Table 2**Sample Characteristics by Exposure Group**

	PCE ¹ n = 55 Mean (SD) or n (%)	OD ² n = 61 Mean (SD) or n (%)	UN ³ n = 23 Mean (SD) or n (%)
Maternal Characteristics			
Race ⁴			
White	23 (41.8%)	39 (63.9%)	6 (26.1%)
Black	18 (32.7%)	12 (19.7%)	8 (34.8%)
Hispanic	11 (20%)	7 (11.5%)	8 (34.8%)
Other	3 (5.5%)	3 (4.9%)	1 (4.3%)
Low SES (Hollingshead 5), 1m	9 (17.6%)	7 (11.9%) ^B	7 (30.4%)
Below poverty line, 1 m	23 (47.9%) ^A	16 (28.6%) ^B	12 (52.2%)
Married	7 (13%) ^{A,B}	27 (44.3%)	10 (43.5%)
Education < high school	23 (41.8%) ^A	13 (21.3%)	8 (34.8%)
No prenatal care	9 (16.4%) ^{A,B}	0 (0%)	0 (0%)
Age	29.5 (4.8)	29.1 (5.9)	28.1 (5.7)
Prenatal drug use (yes)			
Cocaine	55 (100%)	-	-
Opiates	9 (16.4%)	10 (16.4%)	-
Marijuana	17 (30.9%) ^A	3 (4.9%)	-
Alcohol	35 (63.6%)	47 (77%)	-
Nicotine	47 (85.5%) ^A	27 (44.3%)	-
Postnatal Environment			
Postnatal drug use (yes)			
Cocaine	0 (0%)	0 (0%)	0 (0%)
Opiates	0 (0%)	2 (3.3%) ^B	0 (0%)
Marijuana	3 (5.5%)	2 (3.3%)	0 (0%)
Alcohol	23 (41.8%) ^A	44 (72.1%) ^B	8 (34.8%)
Nicotine	26 (47.3%) ^{A,B}	16 (26.2%) ^B	3 (13%)
Domestic Violence, 3–9yr	9 (16.4%)	11 (18%)	1 (4.3%)
Child abuse, 18m–9yr	10 (18.2%)	4 (6.6%)	1 (4.3%)
# of caretaker Δ, 18m–9yr	1.3 (1.3) ^{A,B}	0.16 (0.73)	0 (0)
Newborn Medical Characteristics			
Premature (yes)	21 (38.2%)	18 (29.5%)	6 (26.1%)
Birth weight, g	2640.7 (667.7) ^A	2996.3 (849.9)	2987.5 (904.1)
Length, cm	47.1 (4.1)	48.7 (5.3)	48.7 (5.5)
Head circumference, cm	32.5 (2.5) ^A	33.5 (2.8)	33.3 (3)
Male	32 (58.2%)	31 (50.8%)	12 (52.2%)

¹ Cocaine = exposure to cocaine alone or in combination with other substances;

² Other = other drug exposure without cocaine;

³ No = no exposure to cocaine, opiates, marijuana, alcohol, and/or tobacco;

⁴ Race was identified by mother.

^A Different from the Other Drug (OD) group, $p < .05$;

^B Different from UNexposed (UN) group, $p < .05$

Table 3

Correlations among infant, preschool, and school-age sleep problem indices

	Preschool Sleep Problem Index	School-age Sleep Problem Index
Toddler Sleep Problem Index	.272 [*]	.25 [*]
Preschool Sleep Problem Index		.478 ^{**}

^{*}
 $p < .01$,

^{**}
 $p < .001$