Published in final edited form as:
Arch Pediatr Adolesc Med. 2010 May ; 164(5): 452–456. doi:10.1001/archpediatrics.2010.52.

The Maternal Lifestyle Study: Sleep Problems in Children with Prenatal Substance Exposure

Kristen C. Stone, Ph.D.[*,1], **Linda L. LaGasse, Ph.D.**[1], **Barry M. Lester, Ph.D.**[1], **Seetha Shankaran, MD**[2], **Henrietta S. Bada, MD**[3], **Charles R. Bauer, MD**[4], and **Jane A. Hammond, Ph.D.**[5]

[1]Brown Center for the Study of Children at Risk, Women and Infants' Hospital, Providence, RI

[2]Department of Pediatrics, Wayne State University School of Medicine, Detroit, MI

[3]Department of Pediatrics, University of Kentucky College of Medicine, Lexington, KY

[4]Department of Pediatrics, University of Miami, Miami FL

[5]RTI International, Rockville, MD

Abstract

Objective—To examine the relationships between sleep problems and prenatal exposure to cocaine, opiates, marijuana, alcohol, and nicotine in children 1 month to 12 years of age.

Design—Sleep data was collected by maternal report in a prospective longitudinal follow-up of children participating in the Maternal Lifestyle multisite study.

Setting—Hospital based research centers in Providence, RI, Miami, FL, Detroit, MI, and Memphis, TN

Participants—There were 808 participants: 374 exposed to cocaine and/or opiates; 434 comparison.

Main exposure—Prenatal cocaine, opiate, marijuana, alcohol, and nicotine exposure.

Outcome measure—Sleep problems in early, middle, and late childhood, assessed as composites of maternal report items.

Results—Of the five substances, prenatal nicotine exposure was the only unique predictor of sleep problems (B = .074, R^2 Δ = .008, p = .012) with adjustment for covariates including SES, marital status, physical abuse, prenatal medical care, and postnatal cigarette smoke exposure.

Conclusion—Prenatal exposure to nicotine was positively associated with children's sleep problems persisting throughout the first 12 years of life. Targeting this group of children for educational and behavioral efforts to prevent and treat sleep problems is merited given that good sleep may serve as a protective factor for other developmental outcomes.

Sleep problems in children are associated with daytime impairment including altered psychomotor performance[1], behavioral disturbance[2], sleepiness [3, 4], decreased physical activity and social interest[4], memory and learning deficits[5], and substance use[6]. The role of sleep in the development of children with prenatal drug exposure, however, is not well understood. Sleep studies with prenatally exposed children have been limited almost entirely to infancy. Observable decrements in sleep duration and continuity and in sleep-state

[*]Correspondence: Kristen C. Stone, PhD, Brown Center for the Study of Children at Risk, 101 Dudley Street, Providence, RI 02905; Tel: +1-401-274-1122 ex. 8970; Fax: +1-401-453-7646; kristen_stone@brown.edu.

organization have been found in infants with prenatal cocaine exposure[7-10] and, more recently, in infants with prenatal exposure to alcohol[11] and nicotine[11, 12]. In addition, differences in electroencephalographic sleep patterns between exposed and unexposed infants have been demonstrated in studies of prenatal exposure to opiates[13], alcohol[14], and nicotine[15].

Until recently, there were no longitudinal sleep studies of prenatal exposure to cocaine, opiates, alcohol, or nicotine. One study in 3-year olds found more disrupted sleep related to prenatal exposure to marijuana[16]. Furthermore, an association between sleep problems through 9 years of age and prenatal nicotine exposure has been documented in a small Maternal Lifestyle Study (MLS) sample (n = 139)[17], meriting a larger study with better control of covariates and more power to investigate level of prenatal exposure. The aims of the current study, therefore, were to investigate the effects of prenatal exposure (including level of exposure) to cocaine, opiates, marijuana, nicotine, and alcohol on sleep problems over time (1 month to 12 years) of a large sample of children (n = 808) and to examine the relationship between early sleep problems and later sleep problems.

Patients and Methods

Study Design

The MLS is a multi-site longitudinal investigation of the developmental effects of prenatal exposure to cocaine and other drugs[18-20]. The MLS enrolled 1388 children at birth from 1993 to 1995 and was approved by an appropriate Institutional Review Board at each of its four sites (Wayne State University, Detroit, MI; University of Tennessee at Memphis, Memphis, TN; University of Miami, Miami, FL; Brown University, Providence, RI)[21]. Confidentiality regarding the participants' drug use was assured through each center's NIDA Certificate of Confidentiality.

Mothers were recruited in the hospital after delivery and informed consent was obtained at that time. All women delivering very low birth weight newborns (i.e., 501 to 1500 g) were approached in order to maximize likelihood of recruiting participants that either had prenatal cocaine exposure or were appropriate group-matched controls. During normal business hours other mothers (i.e., those delivering newborns > 1500 g) were also approached. Mothers were eligible who were 18 years or older without psychiatric disorders, developmental delays, or language barriers. Neonates were eligible who were inborn, likely to survive, singleton, and < 43 weeks gestational age. Mother-infant dyads were excluded from the study, starting with the 1-month visit, when the infant had 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. Another match was generated when an infant in the comparison group did not attend the 1-month visit. It was not always possible, however, to replace exposed infants who withdrew, which yielded uneven groups: 658 in the cocaine/opiate-exposed group and 730 in the comparison group.

Participants

Mothers were assigned to the cocaine/opiate exposure group based on either maternal report or meconium toxicology with gas chromatography-mass spectroscopy assay confirmation or both. To be assigned to the comparison group participants who denied cocaine/opiate use during pregnancy also had to have negative meconium toxicology results. The same procedure was followed for identification of marijuana exposure. Alcohol and tobacco use were assessed by maternal report alone. Level (i.e., amount) of prenatal exposure to each of

those five substances (cocaine, opiates, marijuana, alcohol, and nicotine) was determined at the 1-month visit using the Maternal Interview of Substance Use (MISU), which provides information about the frequency (e.g., number of times using cocaine each week) and quantity (i.e., number of cigarettes, joints, and ounces of alcohol used/consumed each day) of substance use during pregnancy. To be eligible for the current study, participants must have completed more than half of all the sleep measures administered from 1 month to 12 years. 808 participants were eligible (374 exposed to cocaine and/or opiates; 434 comparison), which is the final sample for this study.

Demographic and Maternal Measures

At the 1-month visit self-report information about prenatal care, maternal age, marital status, education level, socioeconomic status (SES), and poverty status was gathered. The Caretaker Inventory of Substance Use (CISU) was used at yearly visits to assess level of postnatal use of each of the 5 substances since pregnancy. Furthermore, questionnaires assessed number of caretaker changes (number of times the child changed caregivers over the 12-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 5-12).

Sleep Measures

The sleep problems index was created to measure global sleep onset and maintenance disturbance in our sample across the first 12 years of life using sleep items on the four pediatric questionnaires described below. All items within 3 time periods (i.e., 1 month to 4 years, 5-8 years, and 9-12 years) were summed for 3 age-relative indexes of sleep problems. We then computed a total sleep problem index summing all 45 items from 1 month to 12 years. Our sleep-problem indexes were pro-rated for missing items. That is, we accounted for missing data by multiplying the mean of the items that were present by the total number of possible items. Other studies have created a composite score from questionnaires, such as the CBCL[25, 26], with adequate internal consistency (e.g., alpha = .64)[25]. The Cronbach's alphas for the sleep problems index at each time period for our study were .64, .74, and .8 respectively. The Cronbach's alpha for the overall sleep problems index was .88.

The Child Behavior Checklist (CBCL)[22] was administered at the 3-, 5-, 7-, 9- and 11-year assessments. This standardized parent-report questionnaire identifies behavioral and emotional problems of children over the past 6 months 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 Child Health and Illness Profile was administered at years 10-12. This validated questionnaire [23] assesses children's health and well-being and includes one item assessing “trouble falling asleep.” If endorsed, the child was assigned 1 point. The child was also assigned 1 point if the parent endorsed “trouble sleeping” on the Pediatric Symptom Checklist (PSC), which was administered at years 8-12. The PSC is a 35-item psychosocial screener for cognitive, behavioral, and emotional problems [24].A general health questionnaire administered yearly contained the following sleep items: problems with sleeping, falling asleep at an unreasonable time, waking at night requiring attention, medicated for insomnia, sleep disorder diagnosis. The child was assigned 1 point for each item endorsed by the parent.

Statistical Analyses

Chi-square and *t*-tests were used to compare sample characteristics of attrition groups (Table 1) and exposure groups (Table 2). Analysis of variance (ANOVA) was used to compare the cocaine/opiate exposure group to the comparison group on the overall sleep problem index.

A 2-step approach was used to determine which of the 5 substances (cocaine, opiates, marijuana, alcohol, nicotine) were significant predictors of sleep problems. In step 1, all 5 substances were entered together in a regression model to determine which of the substances reached significance ($p < .05$) suggestive of an association with sleep problems unadjusted for covariates. Step 2 controlled for covariates using hierarchical linear regressions performed with the significant substances from step 1. From the candidate variables listed in Table 2, SES, marital status, abuse (i.e., child was victim of physical or sexual abuse, yes or no), and prenatal medical care (yes or no) were selected as covariates, having met the following criteria: (a) differed significantly between exposure groups, (b) were associated with the overall sleep index at $p \leq .2$, and (c) were not highly correlated with other covariates (i.e., $r > .7$). Additionally, study site and postnatal cigarette smoke exposure were included as covariates, having been selected *a priori*. Pearson correlations were used to investigate the association between early and later sleep problems.

Results

When comparing participants in the 12-year follow-up (i.e., those included in this study) with those not in the 12-year follow-up (Table 1), children in the 12-year follow-up were more likely to be minority (i.e., African American, Hispanic, or Other) and had more caretaker changes over time. Mothers or caregivers in this study were less likely to use marijuana prenatally and more likely to use cocaine, marijuana, and/or alcohol postnatally. There were no significant differences on newborn medical characteristics (i.e., prematurity, birth weight, length at birth, head circumference at birth, gender). There were no significant differences in level (i.e., amount) of postnatal substance use (cocaine, opiates, marijuana, alcohol, or nicotine) among the two attrition groups.

There were also differences between the two exposure groups (Table 2). Compared with comparison mothers (i.e., mothers who did not use cocaine during pregnancy), mothers in the exposed-group were more likely to be low SES, more likely to be older, less likely to be married, less likely to have at least a high school education, and less likely to have had any prenatal care. Furthermore, exposed-group mothers/caregivers were more likely to use substances (cocaine, opiates, marijuana, alcohol, and nicotine) both pre- and postnatally. Compared with comparison children, children in the exposed-group were more likely to have been physically or sexually abused and had more caretaker changes over time.

In the unadjusted analysis among the 5 substances, prenatal nicotine exposure was the only significant predictor of sleep problems ($\beta = .168$, $p < .001$). Importantly, multicollinearity was not a problem as evidenced by tolerance remaining above .7 and the variance inflation factor remaining between 1 and 2. None of the prenatal exposure variables were highly correlated with each other (all *r*'s below .4). When adjusted for covariates (SES, marital status, physical/sexual abuse, prenatal care, clinic site, and postnatal cigarette smoke exposure), the effect of prenatal nicotine exposure predicting more sleep problems on the sleep problem index was still statistically significantly (unstandardized coefficient [B] = .074, $R^2 = .067$, $R^2 \Delta = .008$, $p = .012$). Higher levels of prenatal exposure to nicotine predicted more sleep problems. Specifically, after controlling for covariates, each additional cigarette smoked per day over pregnancy was associated with a .074 increase in the child's sleep problems index score. Increases in this score indicate more sleep onset and maintenance disturbance from 1 month to 12 years.

In a separately tested model, postnatal cigarette smoke exposure was no longer significant when entered into a regression model after prenatal nicotine exposure and covariates; that is, it did not explain sleep problems above and beyond prenatal nicotine exposure and the other covariates.

Discussion

The main finding from this study was that in a large sample of children with prenatal exposure to cocaine, opiates, marijuana, alcohol, and/or nicotine a unique effect on sleep problems between 1 month and 12 years was found for nicotine in both unadjusted and adjusted analyses. This was a dose-response effect for prenatal nicotine exposure with adjustment for covariates. Higher levels of prenatal nicotine exposure predicted more sleep problems, specifically difficulty falling and staying asleep, from 1 month to 12 years. This effect was observed controlling for postnatal maternal/caregiver use of cigarettes.

The finding that prenatal nicotine exposure predicted sleep problems in children is consistent with studies of smokers showing sleep disruption attributable to nicotine[27, 28] and supports a recent study which found that prenatal maternal smoking resulted in less sleep and more fragmented sleep among newborns[12]. Preclinical studies of prenatal nicotine exposure have shown abnormal cardiorespiratory response during sleep[29], altered sleep-state maturation[30], and decreased Rapid Eye Movement (REM) sleep[31]. Also, a recent study documented significantly less sleep in infants after drinking breast milk containing nicotine than after drinking nicotine-free breast milk (53 vs. 85 minutes)[32].

There are several ways through which nicotine may affect sleep. Nicotine has been shown to excite pedunculopontine nucleus cells, which are involved in the modulation of arousal, wake, and REM sleep[31], suppress pontogeniculooccipital spike activity, which influences the initiation and maintenance of REM sleep[33], and inhibit sleep-promoting neurons in the ventrolateral peroptic area[34].

When investigating prenatal exposure to cocaine, opiates, marijuana, alcohol, and nicotine, we found dose response effects of prenatal nicotine exposure only on sleep problems in a large sample of children up to 12 years old. Caution should be taken when concluding that prenatal nicotine exposure is more damaging to sleep compared to other prenatal drug exposure, given the current study's specific limitations (namely the subjectivity of the sleep problems index) and the study's operationalization of “sleep problems” (i.e., difficulty falling and staying asleep). Whether or not there are associations between prenatal drug exposure and other types of sleep problems (e.g., sleep disordered breathing) is unknown and was not investigated in this study. Our study is also limited by the use of maternal self-report for our measures both of sleep problems in the children and of caregiver postnatal substance use. Validated parent-report sleep-problem questionnaires would have been preferable to our index comprised of items from the CBCL, CHIP, PSC, and general health questionnaire. Though this type of validated instrument does not yet exist to measure sleep during the first 12 years of life or to measure pediatric difficulties falling and staying asleep, specifically, this limitation points to an important need in the field. Furthermore, it would be useful to conduct formal sleep studies on these children and validate environmental tobacco exposure and postnatal substance use with toxicological assays.

Conclusion

This is the first longitudinal study of the effects of prenatal drug exposure on sleep problems over time with adjustment for multiple covariates (SES, marital status, physical/sexual abuse, prenatal care, clinic site, and postnatal cigarette smoke exposure) and adequate power

to detect a dose response association. Findings suggest a link between prenatal nicotine exposure and persisting sleep problems in children for the first 12 year of life. Understanding the persisting nature of sleep problems in children with prenatal adversity including substance exposure could be a critical component to improving developmental outcomes in this population. Sleep problems may mediate some of the other developmental effects of prenatal exposure to nicotine. Assessing the risk for sleep problems in children with prenatal exposure to nicotine and other drugs is the first step towards creating efficient, proactive services that will foster optimal sleep and corresponding daytime functioning.

Acknowledgments

This study was supported by the Eunice Kennedy Shriver National Institute of Child Health and Human Development Neonatal Research Network and an interinstitute agreement with the National Institute on Drug Abuse through cooperative agreements U10 DA 024119-01, U10 HD 27904 (to B.M.L.), U10 DA 024117-01, U10 HD 21385 (to S.S.), U10 DA 024128-06, U10 HD 27856 (to H.S.B.), and Child Health and Human Development contract N01-HD-2-3159 (to B.M.L.). Kristen C. Stone, PhD, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

References

1. Anders TF, Keener MA, Kraemer H. Sleep-wake state organization, neonatal assessment and development in premature infants during the first year of life. II. Sleep 1985;8:193–206. [PubMed: 4048735]
2. Thoman EB, Denenberg VH, Sievel J, Zeidner LP, Becker P. State organization in neonates: Developmental inconsistency indicates risk for developmental dysfunction. Neuropediatrics 1981;12:45–54. [PubMed: 6166893]
3. Carskadon, MA. Sleeping and waking disorders: Indications and techniques. Guilleminault, C., editor. Menlo Park, CA: Addison Wesley; 1982. p. 99-125.
4. Carskadon MA, Harvey K, Duke P, Anders TF, Litt IF, Dement WC. Pubertal changes in daytime sleepiness. Sleep 1980;2:453–460. [PubMed: 7403744]
5. Carskadon MA, Acebo C, Jenni OG. Regulation of adolescent sleep: Implications for behavior. Ann N Y Acad Sci Jun;2004 1021:276–291. [PubMed: 15251897]
6. Carskadon MA. Patterns of sleep and sleepiness in adolescents. Pediatrician 1990;17(1):5–12. [PubMed: 2315238]
7. DiPietro JA, Suess PE, Wheeler JS, Smouse PH, Newlin DB. Reactivity and regulation in cocaine-exposed neonates. Infant Behav Dev 1995;18:407–414.
8. Gingras JL, Feibel JB, Dalley LB, Muelenaer A, Knight CG. Maternal polydrug use including cocaine and postnatal infant sleep architecture: preliminary observations and implications for respiratory control and behavior. Early Hum Dev Nov 24;1995 43(3):197–204. [PubMed: 8835189]
9. Regalado MG, Schechtman VL, Del Angel AP, Bean XD. Sleep Disorganization in Cocaine-Exposed Neonates. Infant Behav Dev 1995;18:319–327.
10. Regalado MG, Schechtman VL, Del Angel AP, Bean XD. Cardiac and respiratory patterns during sleep in cocaine-exposed neonates. Early Hum Dev Mar 22;1996 44(3):187–200. [PubMed: 8654312]
11. Troese M, Fukumizu M, Sallinen BJ, et al. Sleep fragmentation and evidence for sleep debt in alcohol-exposed infants. Early Hum Dev Sep;2008 84(9):577–585. [PubMed: 18400423]
12. Stephan-Blanchard E, Telliez F, Leke A, et al. The influence of in utero exposure to smoking on sleep patterns in preterm neonates. Sleep Dec 1;2008 31(12):1683–1689. [PubMed: 19090324]
13. Hutchings DE. Methadone and heroin during pregnancy: a review of behavioral effects in human and animal offspring. Neurobehav Toxicol Teratol Jul-Aug;1982 4(4):429–434. [PubMed: 7121694]
14. Chiriboga CA. Fetal effects. Neurol Clin Aug;1993 11(3):707–728. [PubMed: 8377750]
15. Pichini S, Garcia-Algar O. In utero exposure to smoking and newborn neurobehavior: how to assess neonatal withdrawal syndrome? Ther Drug Monit Jun;2006 28(3):288–290. [PubMed: 16778707]

16. Dahl RE, Scher MS, Williamson DE, Robles N, Day N. A longitudinal study of prenatal marijuana use. Effects on sleep and arousal at age 3 years. Arch Pediatr Adolesc Med Feb;1995 149(2):145–150. [PubMed: 7849875]

17. Stone KC, High PC, Miller-Loncar CL, LaGasse LL, Lester BM. Longitudinal study of maternal report of sleep problems in children with prenatal exposure to cocaine and other drugs. Behav Sleep Med 2009;7:196–207. [PubMed: 19787489]

18. Bada HS, Das A, Bauer CR, et al. Gestational cocaine exposure and intrauterine growth: maternal lifestyle study. Obstet Gynecol Nov;2002 100(5 Pt 1):916–924. [PubMed: 12423853]

19. Bauer CR, Langer JC, Shankaran S, et al. Acute neonatal effects of cocaine exposure during pregnancy. Arch Pediatr Adolesc Med Sep;2005 159(9):824–834. [PubMed: 16143741]

20. Lester BM, El Sohly M, Wright LL, Smeriglio VL, Verter J, Bauer CR. The Maternal Lifestyle Study: Drug use by meconium toxicology and maternal self-report. Pediatrics 2001;107:309–317. [PubMed: 11158464]

21. Lester BM, Tronick EZ, LaGasse L, et al. The Maternal Lifestyle Study: Effects of substance exposure during pregnancy on neurodevelopmental outcome in 1-month-old infants. Pediatrics Dec;2002 110(6):1182–1192. [PubMed: 12456917]

22. Achenbach, TM. Manual for the Child Behavior Checklist/ 4-18. Burlington, Vermont: University of Vermont; 1991.

23. Starfield B, Bergner M, Ensminger M, et al. Adolescent health status measurement: development of the Child Health and Illness Profile. Pediatrics Feb;1993 91(2):430–435. [PubMed: 8424023]

24. Jellinek MS, Murphy JM, Robinson J, Feins A, Lamb S, Fenton T. Pediatric Symptom Checklist: screening school-age children for psychosocial dysfunction. J Pediatr Feb;1988 112(2):201–209. [PubMed: 3339501]

25. Alfano C, Ginsburg G, Kingery J. Sleep-related problems among children and adolescents with anxiety disorders. J Am Acad Child Adolesc Psychiatry 2007;46:224–232. [PubMed: 17242626]

26. Storch EA, Murphy TK, Lack CW, Geffken GR, Jacob ML, Goodman WK. Sleep-related problems in pediatric obsessive-compulsive disorder. J Anxiety Disord 2008;22(5):877–885. [PubMed: 17951025]

27. Patten CA, Choi WS, Gillin JC, Pierce JP. Depressive symptoms and cigarette smoking predict development and persistence of sleep problems in US adolescents. Pediatrics Aug;2000 106(2):E23. [PubMed: 10920179]

28. Zhang L, Samet J, Caffo B, Punjabi NM. Cigarette smoking and nocturnal sleep architecture. Am J Epidemiol Sep 15;2006 164(6):529–537. [PubMed: 16829553]

29. Hafstrom O, Milerad J, Sundell HW. Prenatal nicotine exposure blunts the cardiorespiratory response to hypoxia in lambs. Am J Respir Crit Care Med Dec 15;2002 166(12 Pt 1):1544–1549. [PubMed: 12471072]

30. Frank MG, Srere H, Ledezma C, O'Hara B, Heller HC. Prenatal nicotine alters vigilance states and AchR gene expression in the neonatal rat: implications for SIDS. Am J Physiol Regul Integr Comp Physiol Apr;2001 280(4):R1134–1140. [PubMed: 11247836]

31. Garcia-Rill E, Buchanan R, McKeon K, Skinner RD, Wallace T. Smoking during pregnancy: postnatal effects on arousal and attentional brain systems. Neurotoxicology Sep;2007 28(5):915–923. [PubMed: 17368773]

32. Mennella JA, Yourshaw LM, Morgan LK. Breastfeeding and smoking: short-term effects on infant feeding and sleep. Pediatrics Sep;2007 120(3):497–502. [PubMed: 17766521]

33. Vazquez J, Guzman-Marin R, Salin-Pascual RJ, Drucker-Colin R. Transdermal nicotine on sleep and PGO spikes. Brain Res Oct 21;1996 737(1-2):317–320. [PubMed: 8930383]

34. Saint-Mleux B, Eggermann E, Bisetti A, et al. Nicotinic enhancement of the noradrenergic inhibition of sleep-promoting neurons in the ventrolateral preoptic area. J Neurosci Jan 7;2004 24(1):63–67. [PubMed: 14715938]

Table 1

Sample Characteristics by Attrition

	With 12yr data* n = 808 (58.2%)	Without 12yr data* n = 580 (41.8%)	*p*
Maternal Characteristics			
Race, minority	689 (86.4%)	462 (80.5%)	.003
Low SES (Hollinghead 5), 1mo.	193 (24.5%)	116 (21.2%)	.163
Below poverty line, 1 mo.	484 (64.5%)	325 (62.6%)	.486
Married	144 (17.8%)	122 (21.1%)	.128
Education < high school	311 (38.5%)	234 (40.5%)	.465
No prenatal care	89 (11%)	79 (13.6%)	.142
Age	28.6 (5.8)	28 (5.9)	.106
Prenatal drug use (yes)			
Cocaine	346 (42.8%)	254 (43.8%)	.719
Opiates	58 (7.2%)	57 (9.8%)	.077
Marijuana	173 (21.4%)	151 (26%)	.045
Alcohol	487(60.3%)	338 (58.3%)	.455
Nicotine	422 (52.2%)	326 (56.2%)	.142
Postnatal Environment			
Postnatal drug use (yes)			
Cocaine[1]	102 (12.6%)	46 (8.6%)	.022
Opiates	32 (4%)	26 (4.9%)	.419
Marijuana	215 (26.6%)	111 (20.8%)	.016
Alcohol	667 (82.5%)	389 (73%)	< .001
Nicotine	519 (64.2%)	339 (63.6%)	.814
Domestic Violence, 5-12yr	108 (13.5%)	45 (17.9%)	.084
Child abuse, 1mo-12yr,	204 (25.2%)	128 22.4 %	.219
# of caretaker Δ, 4mo-12yr	1(1.6)	0.8 (1.2)	.004
Newborn Medical Characteristics			
Premature (yes)	339 (42.1%)	238 (41.1%)	.722
Birth weight, g	2615.4 (833.8)	2649.9 (797.1)	.439
Length, cm	46.6 (5.1)	46.9 (4.9)	.346
Head circumference, cm	32.1 (3)	32.2 (3)	.439
Male	427 (52.8%)	300 (51.7%)	.68

*% or Mean (SD)

[1]The decrease in cocaine use postnatally may be attributable to the fact that mothers using cocaine during pregnancy in some cohorts of the MLS (e. g., Providence) were required by state law to remain abstinent from cocaine in order to be reunified with their children. Either the mother remained abstinent from cocaine or the child was raised by a caregiver that reported abstinence from cocaine. Admitting postnatal cocaine use 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 the participant's drug use. The certificate superseded any mandatory reporting of illegal substance use and was explained in full to the mothers.

Table 2

Sample Characteristics by Original MLS Exposure Groups

	Exposure*[1] n = 374 (46.3%)	**Comparison* n = 434 (53.7%)**	***p***
Maternal Characteristics			
Race, minority	319 (86.9%)	370 (86%)	.719
Low SES (Hollinghead 5), 1mo	100 (28.1%)	93 (21.6%)	.035
Below poverty line, 1 mo	232 (68.2%)	252 (61.5%)	.054
Married	36 (9.7%)	108 (24.9%)	< .001
Education < high school	197 (52.8%)	299 (68.9%)	< .001
No prenatal care	79 (21.1%)	10 (2.3%)	< .001
Age	30.42 (4.98)	26.96 (5.93)	< .001
Prenatal drug use (yes)			
Cocaine	346 (92.5%)	0 (0%)	< .001
Opiates	58 (15.5%)	0 (0%)	< .001
Marijuana	138 (36.9%)	35 (8.1%)	< .001
Alcohol	270 (72.2%)	217(50%)	< .001
Nicotine	300 (80.2%)	122(%)	< .001
Postnatal Environment			
Postnatal drug use (yes)			
Cocaine[2]	95 (25.4%)	7 (1.6%)	< .001
Opiates	29 (7.8%)	3 (.7%)	< .001
Marijuana	130 (34.8%)	85 (19.6%)	< .001
Alcohol	326 (87.2%)	341 (78.6%)	< .001
Nicotine	314 (84%)	205 (47.2%)	< .001
Domestic Violence, 5-12yr	56 (15.2%)	52 (12%)	.19
Child abuse, 1mo-12yr,	118 (31.6%)	86 (19.8 %)	< .001
# of caretaker Δ, 4 mo-12yr	1.64 (1.82)	.39 (1)	< .001
Newborn Medical Characteristics			
Premature (yes)	159 (42.6%)	180 (41.6%)	.762
Birth weight, g	2,568.24 (776.85)	2,656.05 (878.74)	.132
Length, cm	46.43 (4.81)	46.82 (5.3)	.277
Head circumference, cm	31.97 (2.81)	32.14 (3.23)	.432
Male	173 (46.3%)	208 (47.9%)	.635
Sleep Problems Index			
Sleep Problems Index, 1m-12yr	6.67 (5.65)	6.5 (5.78)	.673

*% or Mean (SD)

[1]Participants were recruited to be in 1 of 2 groups: the group with exposure to cocaine or opiates or the comparison group that was matched on ethnicity, gender, and gestational age.

[2]The decrease in cocaine use postnatally may be attributable to the fact that mothers using cocaine during pregnancy in some cohorts of the MLS (e. g., Providence) were required by state law to remain abstinent from cocaine in order to be reunified with their children. Either the mother remained abstinent from cocaine or the child was raised by a caregiver that reported abstinence from cocaine. Admitting postnatal cocaine use 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 the participant's drug use. The certificate superseded any mandatory reporting of illegal substance use and was explained in full to the mothers.