Published in final edited form as:
Infant Ment Health J. 2006 ; 27(4): 383–404. doi:10.1002/imhj.20097.

Prenatal Cocaine Exposure and Infant Sleep at 7 Months of age: The Influence of the Caregiving Environment

Pamela Schuetze,
SUNY at Buffalo

Desirae Lawton, and
SUNY College at Buffalo

Rina D. Eiden
SUNY at Buffalo

Abstract

The primary goal of this study was to examine sleep problems in a sample of cocaine-exposed 7-month-old infants and to determine if maternal psychopathology mediated any existing association between substance exposure and sleep behaviors. We also examined the differences in sleep behaviors of cocaine-exposed infants in parental custody and cocaine-exposed infants in nonparental custody. Participants were 65 cocaine-exposed and 53 nonexposed infants and their primary caregivers who were recruited at delivery and assessed at 7 months of infant age. As expected, women who used cocaine during pregnancy had more psychiatric symptoms than nonusers. Prenatal exposure to heavier amounts of cocaine was significantly related to more severe sleep difficulties, and maternal anxiety mediated this association. Approximately 28% of cocaine mothers lost custody of their infants by 7 months of age. Nonmaternal caregivers had significantly fewer symptoms of psychopathology than the cocaine-using women who retained custody of their children. Infants who were in nonparental care at 7 months of age also had less severe sleep problems than did infants who remained in parental care.

Prenatal exposure to cocaine has been associated with altered postnatal behavior, particularly in the realm of arousal and behavioral organization. Cocaine has been shown to inhibit the reuptake of monoamines at the presynaptic junction, leading to higher concentrations of norepinephrine, serotonin, and dopamine in the synaptic cleft and higher levels of activation in the catecholaminergic systems (Gawin & Ellinwood, 1988; Nassogne, Evrard, & Courtoy, 1998). The regions of the brain that are rich in monoamines are the very centers involved in regulatory activities and reactivity to stress (Robbins, 1997; Tucker & Williamson, 1984). In fact, a growing body of evidence has suggested that prenatal exposure to cocaine may have a significant impact on the development of regulatory processes in infants. These regulatory difficulties include poorer state regulation, more difficulty being calmed, increased physiological arousal, and differential physiological responding to sensory challenges, as measured by both heart rate and cortisol levels, beginning in the neonatal period and persisting through-out the first year of life (Bendersky & Lewis, 1998a, 1998b; Brown, Bakeman, & Coles, 1998; DiPietro, Suess, Wheeler, Smouse, & Newlin, 1995; Gingras, Feibel, Dalley, Muelenaer, & Knight, 1992; Karmel & Gardner, 1996; Mayes, Bornstein, Chawarska, & Granger, 1995, 1996; Regalado, Schechtman, Del Angel, & Bean, 1995, 1996; Schuetze & Eiden, 2006).

Direct correspondence to: Pamela Schuetze, Department of Psychology, SUNY College at Buffalo, 1300 Elmwood Avenue, Buffalo, NY 14222–1095; schuetp@buffalostate.edu.

To date, the majority of studies examining regulatory processes among cocaine-exposed infants have focused on behaviors that occur during waking periods; however, regulatory processes during the first year of life include the modulation of behavioral states in periods of both wakefulness and sleep (see Bornstein & Lamb, 1992). Despite this, relatively few studies have examined aspects of sleep in this population. There is evidence to suggest that cocaine-exposed neonates have altered sleep patterns including less time spent in sleeping states, decreased active sleep, and more frequent arousals during active sleep (DiPietro et al., 1995; Gingras, Feibel, Dalley, Muelenaer, & Knight, 1995; Regalado et al., 1995). Furthermore, sleep disruptions have been noted among neonates prenatally exposed to other drugs including alcohol (Scher, Richardson, Coble, Day, & Stoffer, 1988), marijuana (Scher et al., 1988), and opiates (Dinges, Davis, & Glass, 1980). Since maternal cocaine use during pregnancy is highly associated with the use of other substances, there is reason to suspect a higher incidence of sleep problems among cocaine-exposed infants. However, all of these studies were conducted with neonates. Thus, it is unclear if neonatal sleep disturbances would persist causing sleep problems later in infancy. One recent study found that infants who had been prenatally exposed to cocaine had lower spectral electroencephalographic (EEG) power values at 1 year of age, but it was unclear if these altered quantitative EEG measures would translate to altered sleep behaviors (Scher, Richardson, & Day, 2000); however, these results do highlight the importance of examining sleep in older cocaine-exposed infants. Furthermore, sleep difficulties, which include resisting going to bed and/or settling down to sleep and night waking, occur in up to 30% of children in the second half of the first year of life (Lozoff, Wolf, & Davis, 1985; Moore & Ucko, 1957; Richman, 1981). Although night waking is considered normative during the first 6 months of life (Adair, Bauchner, Philipp, Levenson, & Zuckerman, 1991), it is frequently viewed as being very disruptive to family life after that point and is one of the most common problems reported to pediatricians by parents (Anders & Keener, 1985). Infant sleep problems also are associated with both maternal fatigue and child abuse (Chavin & Tinson, 1980), which may have implications for continued social and emotional development. Thus, it is important to examine the presence of sleep problems beyond the neonatal period into later infancy.

In addition to prenatal substance exposure, other factors have been associated with sleep difficulties during infancy, including perinatal problems (Bernal, 1973; Blurton Jones, Rosseti Ferreira, Farguhar Brown, & MacDonald, 1978) and temperament (Halpern, Anders, Garcia Coll, & Hua, 1994; Weissbluth, 1981). Since development is dependent upon an interaction of both biological and environmental influences (Sameroff & Chandler, 1975), it has been hypothesized that the quality of caregiving as well as the sensitivity of the caregiver may contribute to the development or persistence of altered regulatory processes including sleep difficulties (Benoit, Zeanah, Boucher, & Minde, 1992). For example, infant sleep is particularly sensitive to dyadic interactions between infant and caregiver (Anders, 1994). In addition, sleep problems among infants have been associated with an insecure attachment history of the mother as assessed by the Adult Attachment Inventory (Benoit et al., 1992) as well as with parental overconcern (Paret, 1983). Other maternal characteristics such as psychopathology also may impact sleeping behaviors. To date, both maternal depression and family stress have been associated with sleep difficulties (Kataria, Swanson, & Trevarthan, 1987; Richman, 1981; Seifer, Sameroff, Dickstein, Hayden, & Schiller, 1996; Zuckerman, Stevenson, & Bailey, 1987). Maternal cocaine use is associated with a higher risk for comorbid psychopathology such as depression and higher levels of general psychological distress (Boyd, 1993; Eiden, Peterson, & Coleman, 1999; Luthar, Cushing, Merikangas, & Rounsaville, 1998; Singer et al., 1997). These aspects of psychological functioning have consistently been linked to nonoptimal mother–infant interactions and developmental outcomes among women who do not use cocaine during pregnancy (e.g., Dickstein et al., 1998; Field, 1992; Gelfand & Teti, 1990) as well as among mothers who use cocaine and other substances (e.g., Beckwith, Howard, Espinosa, & Tyler, 1999; Luthar

et al., 1998; Singer et al., 1997). Thus, maternal psychopathology may mediate an association between prenatal exposure to cocaine and infant sleep problems.

Taken together, studies of the association between maternal characteristics and infant sleep have suggested that the quality of the caregiving environment is an important influence on sleeping behaviors in the first year of life. At the same time, there is an increasing recognition that the quality of the caregiving environment of cocaine-exposed infants is compromised and that this may exert a significant effect on the quality of child outcomes (Brown, Bakeman, Coles, Platzman, & Lunch, 2004; Hans, 2002; Kettinger, Nair, & Schuler, 2000). Furthermore, a relatively large proportion of cocaine-exposed infants are cared for by caregivers other than their parents. Wasserman and Leventhal (1993) reported that 20% of cocaine-exposed children were in nonparental care compared to 2% of a matched comparison group by 2 years of life. With a few exceptions, results from most studies have indicated that cocaine-exposed children placed in nonparental care have better cognitive and behavioral outcomes compared to those in parental care (e.g., Brown et al., 2004); however, it is unclear if this also would be the case for infant sleep. Consequently, it is important to compare the sleep behaviors of cocaine-exposed infants who remain in parental care to the sleep behaviors of cocaine-exposed infants who are in nonparental care.

Thus, the purpose of the present study was to examine the association between maternal cocaine and other substance use during pregnancy, and postnatal caregiving status on infant sleep behaviors and maternal cognitions about their infants' sleep behaviors. We hypothesized that mothers who had used higher amounts of cocaine during pregnancy would have infants with more severe sleep problems and would have more negative cognitions about their infants' sleep behaviors at 7 months of age. We also hypothesized that maternal psychopathology would be associated with maternal reports of infant sleep behaviors and with maternal cognitions of infant sleep behaviors, and that it would mediate the association between maternal substance use during pregnancy and infant sleep behaviors and maternal cognitions of infant sleep behaviors. Finally, we hypothesized that cocaine-exposed infants who remained in parental care at 7 months would have more sleep problems than would either nonexposed infants or cocaine-exposed infants in nonparental care at 7 months.

I. Method

A. Participants

Participants consisted of 118 mother–infant dyads (65 cocaine-exposed, 53 non-cocaine-exposed) recruited into an ongoing longitudinal study of maternal substance use and child development who completed the 7-month assessment. By 7 months of infant age, 18 infants (28% of those exposed to cocaine) had been removed from parental care and placed in nonparental care. Of these 18 infants, 12 (67%) had been placed in nonparental care by 1 month of child age. The remaining 6 were placed in nonparental care between 1 and 7 months. Approximately 72% of these infants were in non-kin care, with the remainder being cared for by a grandmother or a maternal aunt. Only 1 infant in the noncocaine group was placed in nonparental care and was not included in data analyses. All 7-month assessments were conducted with the primary caregiver of the child at that time.

An outreach worker on the project staff recruited all participants after delivery from two local area hospitals. Mothers ranged in age from 18 to 42 years (M=30.74, SD=5.45). The majority of mothers were African American (61%), were receiving Aid to Families with Dependent Children (71%) at the time of their first laboratory visit (Years 2001–2004), and were single (60%). The two groups were matched on maternal education, maternal age, maternal race/ethnicity, and infant gender. Forty-eight percent of the infants were male. As shown in Table 1, comparison infants had a significantly longer gestation, and increased

birth weight, birth length, and head circumference than did cocaine-exposed infants. Eighty-four percent of the cocaine-exposed and 96% of the comparison infants were full-term (>37 weeks' gestational age). The study received approval from the institutional review boards of the hospitals as well as the primary institutions with which the authors are affiliated. In addition, informed written consent was obtained from all recruited participants. Participants received $35 in monetary incentives at the 4- to 8-week visit and $50 in the form of gift certificates, checks, and infant toys at the 7-month visit for their participation.

B. Procedure

All mothers were approached by study staff at two local hospitals and were invited to participate in a study of maternal health and infant development. In the circumstance of a change in custody arrangements, the person who had legal guardianship of the child was contacted and asked to participate in addition to the biological mother. Interested and eligible mothers and caregivers were given detailed information about the study and asked to sign consent forms. A total of 103 eligible mothers declined to participate. Mothers who participated in the study were more likely to be between 18 and 25 years, $\chi^2(2) = 19.15$, $p < .001$, and to have a high-school or below education, $\chi^2(2) = 45.79$, $p<.001$, than were mothers who declined to participate in the study. There were no other demographic differences between mothers who participated and eligible mothers who declined to participate in the study. Furthermore, there were no differences in gestational age or fetal-growth measures between infants of mothers who participated or declined to participate in the study. About 2 weeks after delivery, caregiver–infant dyads were contacted and scheduled for their first laboratory visit, which took place when the infant was approximately 4 to 8 weeks old. This visit consisted of a caregiver interview, a feeding session, and infant and maternal physiological assessments. A second laboratory visit was scheduled when the infant was about 7 months old. This visit consisted of a caregiver interview, feeding and play sessions that were videotaped, and behavioral and physiological assessments of infant reactivity and regulation. Of the 134 mother–infant dyads who completed the 4- to 8-week laboratory visit, 3 declined to participate in the 7-month assessment, 10 were unable to be located, and 3 mothers no longer had custody of their children and the foster parent was uninterested in participating. Only the data from the caregiver interview and the physiological assessments of infants at the two time points were used in this study.

C. Assessment of Growth and Risk Status

Three measures of growth were used in this study: birth weight (g), birth length (cm), and head circumference (cm). All measurements were taken by obstetrical nurses in the delivery room and recorded in the infant's medical chart. Research staff recorded this information from the charts after recruiting the mother–infant dyad. Medical chart review at the time of recruitment also was used to complete the Obstetrical Complications Scale (OCS; Littman & Parmelee, 1978), a scale designed to assess the number of perinatal risk factors experienced by the infant. Higher numbers on this scale indicate a more optimal obstetric score.

D. Identification of Substance Use

Cocaine status was determined by a combination of maternal report, chart review, and maternal hair analysis. Urine toxicologies were routinely conducted at the first prenatal visit on maternal urine and/or at delivery (for those mothers who tested positive prenatally, obtained prenatal care elsewhere, or did not receive any prenatal care) on infant and maternal urine by participating hospitals. Mothers were included in the cocaine group if self-reports were positive, regardless of urine toxicology or hair-sample results. Similarly, mothers who reported that they did not use cocaine but had positive urine toxicology or hair samples were included in the cocaine group.

Urine toxicologies consisted of standard urine screening for drug level or metabolites of cocaine, opiates, benzodiazepines, and tetrahydrocannabinol. Urine was rated positive if the quantity of drug or metabolite was >300 g/ml. Hair samples were collected from the mothers at the first laboratory visit and sent to the Psychemedics Corporation for radioimmunoanalyses (RIAH). Hair samples were screened for cocaine followed by a gas chromatography/mass spectrometry (GC/MS) confirmation for positive cocaine screens. Drugs and their metabolites are absorbed into the hair and can be extracted and measured. As hair grows at an average rate of 1/2 inch per month, it can record a pattern of drug consumption related to the amount and frequency of use (see Baumgartner, Hill, & Blahd, 1989). Thus, a 2-inch length of hair could contain a record of approximately 4 months of use, and given adequate hair length (i.e., about 4–5 inch), use per trimester may be recorded. Drugs become detectable in hair about 3 to 4 days after use, a time when cocaine is rendered undetectable by urinalysis. RIAH is the most well-established hair-analysis technique and has been replicated by independent laboratories across the world (see Magura, Freeman, Siddiqi, & Lipton, 1992). GC/MS confirmations of RIAH have not revealed any false positives because of testing errors (see Magura et al., 1992).

Approximately 32% of mothers in the study (55% of the mothers in the cocaine group) had positive urine toxicologies at delivery, and 25% of mothers (79% of the mothers in the cocaine group) had hair samples that tested positive for cocaine during pregnancy. The remainder of mothers in the cocaine group admitted having used cocaine in the brief self-report screening instrument administered after delivery. Mothers in the comparison group reported not having used any illicit substances other than marijuana. Additional exclusionary criteria for all mothers were (a) maternal age of 18 years or older, (b) use of illicit substances other than cocaine or marijuana, and (c) significant medical problems for the infant (e.g., genetic disorders, major perinatal complications, baby in critical care for over 48 hr).

The Timeline Follow-Back Interview (TLFB; Sobell, Sobell, Klajner, Pavan, & Basian, 1986) was used to assess maternal substance use before, during, and after pregnancy at the 1-month visit. Participants were provided a calendar and asked to identify events of personal interest (i.e., holidays, birthdays, vacations, etc.) as anchor points to aid recall. This method has been established as a reliable and valid method of obtaining longitudinal data on substance-use patterns, has good test-retest reliability, and is highly correlated with other intensive self-report measures (Brandon, Copeland, & Saper, 1995; Brown et al., 1998). The TLFB yielded data about the number of days cocaine was used, the total number of joints smoked, total number of cigarettes smoked, and total number of standard drinks, mean standard drinks per drinking day, and number of alcohol binges (i.e., >5 standard drinks) for each trimester of pregnancy and for the postnatal period.

Data obtained from both the TLFB interview on cocaine use during pregnancy and from maternal hair analysis were used to identify women as light, heavy, or noncocaine users. Consistent with other studies (e.g., Jacobson et al., 1996), heavy cocaine use was defined as use of cocaine 2 or more days per week during the first trimester of pregnancy or the top quartile of hair-analysis results among the cocaine users (>304.2 ng/10 mg of hair). Mothers who used less cocaine during pregnancy were assigned to the light-cocaine-use group (see Table 1 for information about other substance use in these groups).

E. Caregiver Psychopathology

The Brief Symptom Inventory (BSI; Derogatis, 1993) is a brief form of Symptom Checklist 90-R and is a widely used mental health screening measure in a variety of clinical and research settings. It consists of 53 items rated on a 5-point scale ranging from 0 (never) to 4 (always) and was administered at the 7-month interview. The items are grouped into nine

scales of Anxiety, Hostility, Somatization, Obsessive-Compulsive, Interpersonal Sensitivity, Depression, Phobic Anxiety, Paranoid Ideation, and Psychoticism. These subscales have been reported to have high internal consistency and have been used in a large number of studies, including studies of maternal cocaine use (e.g., Eiden, Lewis, Croff, & Young, 2002; Singer et al., 1997).

F. Infant Sleep Problems

The Maternal Cognitions About Infant Sleep (MCISQ; Morrell, 1999b) is a five-subscale, 27-item questionnaire that assesses mothers' cognitions about their infants' sleep behaviors. Each item is rated on a 6-point Likert-type rating scale ranging from 0 (*strongly agree*) to 5 (*strongly disagree*). This questionnaire has been found to be both reliable and have convergent validity with other measures of infant sleep problems, including sleep diaries (Morrell, 1999b). The five subscales are Limit Setting, Anger, Doubt, Feeding, and Safety. High scores on the Limit Setting subscale indicate difficulty by the parent in resisting infant demands at night. This scale had low internal consistency (Cronbach's α=.58) and was not considered in further analyses. High scores on the Anger subscale reflect feelings of anger, regret, and helplessness that the mother may experience in response to the demands of parenting when the infant wakes or has trouble settling at night. This scale had high internal consistency (Cronbach's α=.83). High scores on the Doubt subscale indicate uncertainty experienced by the mother regarding the adequacy of her parenting as related to infant sleep problems. This scale had moderate internal consistency (Cronbach's α=.75). High scores on the Feeding subscale suggest that the mother believes that feeding is important to soothe her infant at night. This scale had moderate internal consistency (Cronbach's α=.68). Finally, high scores on the Safety subscale indicate more concerns about the possibility of Sudden Infant Death Syndrome (SIDS) occurring while the infant sleeps at night. This scale had moderate internal consistency (Cronbach's α=.67).

The Infant Sleep Questionnaire (ISQ; Morrell, 1999a) is a 10-item questionnaire that assesses infant sleeping habits and parental strategies for managing infant sleep and has been demonstrated to have concurrent validity with other measures of infant sleep problems (Morrell, 1999a). Higher scores indicate more severe sleep problems. The internal consistency for this scale was .77 (Cronbach's α).

G. Infant Physiological Assessment During Rest

Infant physiological regulation during a period of sleep was assessed to examine the association between physiological regulation and maternal reports of infant sleep. Thus, measures of infant heart rate and respiratory sinus arrhythmia (RSA) were obtained during rest at both the 4- to 8-week and 7-month laboratory visits. At 4 to 8 weeks of age, infants were tested in a quiet examining room while in a sleep state. Following a 5-min period of undisturbed acclimation to the physiological acquisition equipment and bassinet in which testing occurred, 10 min of undisturbed physiological data were recorded online directly into a data-acquisition computer. At 7 months of age, infants were tested while seated in a highchair. A resting state was induced by having the infant watch a 5-min segment of a neutral videotape (“Discover Spot” [Buena Vista Home Entertainment, 2000]; see Calkins, 1997, for similar procedures for inducing rest).

Examiners were blind to infant group status. A five-channel Bioamp (James Long Company) recorded respiration and electrocardiograph data. Disposable electrodes were triangulated on the infant's chest. A respiration bellows was placed at the height of the zyphoid process to measure inspiration and expiration.

IBI Analysis software (James Long Company, 1999) was used to process the heart rate data and to calculate RSA. Heart rate samples, which were collected every 10 ms, were used to calculate mean HR per 1-s period. A level detector was triggered at the peak of each R-wave. The interval between sequential R-waves was calculated to the nearest millisecond. Data files of R-wave intervals were manually edited later to remove incorrect detection of the R-wave or movement artifacts. The software computes RSA using respiration and interbeat interval (IBI) data as suggested by Grossman (1983). The difference between maximum IBI during expiration and minimum IBI during inspiration was calculated. The difference, which is measured in seconds, is considered to be a measure of RSA and is measured twice for each respiration cycle (i.e., once for each inspiration and once for each expiration). The time for inspirations and expirations is assigned as the midpoint for each. The time for each arrhythmia sample is assigned as the midpoint between an inspiration time and an expiration time. The software synchronizes with respiration and thus is relatively insensitive to arrhythmia due to tonic shifts in heart rate, thermoregulation, and baroreceptor. Average RSA and heart rate variables were calculated for the 10-min period of sleep at the 4- to 8-week visit and for the 5-min period of rest at the 7-month visit. Decreased RSA is thought to index a dysregulated affective style (Beauchaine, 2002).

II. Results

A. Assessment of Covariates

We first examined the association between substance use, psychopathology, and sleep variables (see Table 2). Both number of binge-drinking episodes and the total number of standard drinks consumed during pregnancy were positively associated with scores on the MCISQ Feeding subscale. Thus, binge drinking and total number of standard drinks consumed during pregnancy were both used as covariates in hierarchical regression analyses examining group differences in MCISQ scores. No other associations between substance use during pregnancy and sleep measures were found.

Next, we examined the relation between maternal psychopathology and demographics and the sleep variables (see Table 2). Scores on the BSI Anxiety and Phobic Anxiety subscales were marginally associated with scores on the ISQ. In addition, parity was marginally associated with scores on the ISQ. Thus, parity was used as a covariate in hierarchical regression analyses examining group differences in ISQ scores. The results using parity as a covariate were no different than when conducting the analyses without parity as a covariate. Consequently, all regression analyses were conducted without parity included as a covariate.

Gender differences for the ISQ and MCISQ measures also were examined. No gender differences were found for any of these measures. Thus, infant gender was not used as a covariate in analyses of group comparisons.

B. Level of Exposure

1. Sample Characteristics—Group differences for the demographic, substance use, and obstetric risk status variables for the three exposure groups are presented in Table 1. Mothers who used lighter amounts of cocaine during pregnancy were significantly older and had experienced more live births than mothers who did not use cocaine during pregnancy. Infants who were exposed to heavier amounts of cocaine during pregnancy had significantly smaller birth weights than did infants in the nonexposed or light-exposure groups. In addition, infants in the light-exposure group had significantly smaller birth weights than did nonexposed infants. Infants in the heavy-exposure group also had significantly shorter birth lengths than did infants in the other two groups and had more optimal obstetrical risk status scores, as measured by the OCS, than did infants in either of the two exposure groups.

Infants who were prenatally exposed to heavy amounts of cocaine had more sleep problems than did nonexposed infants or infants exposed to lighter amounts of cocaine. In addition, mothers who used heavier amounts of cocaine during pregnancy received higher scores on the MCISQ Doubt subscale than did mothers in the other two groups; however, after controlling for alcohol use during pregnancy, group differences for the MCISQ Doubt subscale were not found.

2. Mediational Analyses—The next step was to examine if there was an indirect association between cocaine exposure and infant sleep via maternal psychopathology. Two approaches to examining indirect or mediational pathways have been discussed in recent years (e.g., MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). The first is the widely used causal steps approach to mediation that clearly specify that all independent variables (IV), dependent variables (DV), and mediator variables must be associated with each other to test mediation (Baron & Kenny, 1986; Judd & Kenny, 1981). The shortcomings of this method have been discussed, with three primary ones being that (a) they do not provide a statistical test of the indirect effect of an IV on a DV via a third variable, (b) that large sample sizes (n = > 500) are required to have adequate power to test mediational effects with small to medium effect sizes, (c) and that the condition that IV and DV have to be significantly associated with each other excludes many “inconsistent” intervening-variable models in which the direct and indirect effects have opposite signs and may cancel each other (MacKinnon, Krull, & Lockwood, 2000). Given our more moderate sample size, we chose to analyze the role of maternal psychopathology using an intervening-variable approach (see MacKinnon et al., 2000; MacKimnnon et al., 2002).

Only two BSI subscales, Anxiety and Phobic Anxiety, were associated with both level of cocaine use during pregnancy and infant sleep. Because of the high correlations between these two subscales, $r = .56$, $p < .01$, we only explored the possibility that Anxiety would mediate the relationship between cocaine exposure and scores on the ISQ. Cocaine-group status was dummy coded (i.e., no cocaine vs. light or heavy use and no or light cocaine vs. heavy use). The first step in this process was to estimate the association between maternal cocaine use and scores on the BSI Anxiety subscale using linear regression, with the score on the BSI Anxiety subscale as the criterion variable and maternal cocaine use as the predictor (see Table 3). These analyses indicated that maternal cocaine use was significantly associated with Anxiety. In the next step, the association between Anxiety and scores on the ISQ was estimated. Hierarchical linear regression was used, with the ISQ score as the criterion variable. The Product of Coefficients Test for the intervening variable effect was used to calculate the significance of the indirect effect (see MacKinnon et al., 2002). The standard errors of the intervening-variable effect were calculated with the formula suggested by MacKinnon (1994). The significance of the intervening-variable effect was tested by dividing the estimate of the intervening-variable effect by its standard error, which was then compared to the values of the normal distribution (MacKinnon et al., 2002). The intervening-variable effect for Anxiety when comparing heavy-cocaine-using mothers to nonusing mothers was significant, $z = 2.46$, $p < .05$. Thus, mothers who used heavier amounts of cocaine during pregnancy had more symptoms of anxiety, and mothers with higher levels of anxiety had infants with more reported sleep problems. None of the other scores on the BSI subscales met criteria for an intervening-variable effect with ISQ as the criterion variable.

The same procedure was followed to examine maternal psychopathology as potential intervening variables of the association between maternal cocaine use and maternal cognitions of infant sleep at 7 months of age. None of the measures of maternal psychopathology met criteria for an intervening-variable effect with MCISQ as the criterion variable.

3. Exploratory Analyses of the Association Between Sleep Problems and Physiological Measures—Correlational analyses were conducted to determine whether maternal reports of sleep problems were associated with observed measures of infant physiological regulation during rest at 4 to 8 weeks and at 7 months of infant age. These analyses indicated that scores on the MCISQ subscales and ISQ were related to RSA and heart rate at 4 to 8 weeks and to RSA at 7 months (see Table 4).

C. Parental Versus Nonparental Custody

1. Sample Characteristics—Within the cocaine group, those who retained custody of their children were older and had more prenatal visits compared to those who did not have custody of their children (see Table 3). As expected, mothers with children in nonparental care were heavier users of cocaine compared to cocaine-using mothers who retained custody of their children. Marijuana use also tended to be more frequent among cocaine-using mothers with children in nonparental care compared to those who had custody of their children. Although there were no group differences in alcohol use among the two groups of cocaine-using women, note that the variability in amount of alcohol use was quite large. Cocaine-exposed children in parental care had significantly more sleep problems, as measured by the ISQ, than did cocaine-exposed children in nonparental care (see Table 3). Finally, cocaine-using mothers with custody of their infants had higher scores on the BSI Anxiety, Phobic Anxiety, Obsessive-Compulsive, Interpersonal Sensitivity, Depression, and Paranoid Ideation subscales than did either mothers who used cocaine and did not retain custody of their children or mothers who did not use cocaine during pregnancy (see Table 3).

2. Mediational Analyses—Using the aforementioned procedure, we examined measures of maternal psychopathology as potential intervening variables of the association between caregiving status and infant sleep problems (see Table 5). The first step in this process was to estimate the association between caregiving status and scores on the BSI using separate linear regression analyses. The BSI subscale scores were used as the criterion variables, and caregiving status was the predictor. These analyses indicated that caregiving status was significantly associated with Anxiety, Obsessive-Compulsive, Interpersonal Sensitivity, Depression, Paranoid Ideation, and Hostility; however, none of these variables met criteria for an intervening-variable effect with total score on the ISQ as the criterion variable. Similarly, none of the psychopathology measures met criteria for an intervening-variable effect with either the total score or subscales scores of the MCISQ.

III. Discussion

The primary goal of this study was to examine the level of sleep problems in a sample of cocaine-exposed 7-month-old infants and to determine if maternal psychopathology mediated any existing association. We found that prenatal exposure to heavier amounts of cocaine was significantly related to more severe sleep difficulties. These results indicate that the disorganized-state regulation noted in cocaine-exposed neonates (DiPietro et al., 1995; Hume, O'Donnell, Stanger, Killam, & Gingras, 1989) is still present by 7 months of age. Since increased sleep problems are disruptive to family life (Richman, Douglas, Hung, Lansdown, & Levere, 1985) and are associated with both maternal fatigue and child abuse (Chavin & Tinson, 1980), these findings suggest that the heavily cocaine-exposed mother–infant dyads are at an increased risk for long-term emotional and behavioral problems. Future studies should examine sleep problems in cocaine-exposed children into the preschool years to see if the sleep difficulties persist and if they are indeed associated with nonoptimal developmental outcomes.

We also found that levels of maternal anxiety mediated the association between cocaine exposure and sleep difficulties. Specifically, exposed infants who had mothers with higher levels of anxiety at 7 months of infant age showed higher levels of sleep problems. Given the correlational nature of these findings, it is not clear if this is a causal pathway. One explanation for these findings is that although dysregulated behavioral states during the neonatal period seem to reflect disrupted autonomic nervous system development, the postnatal environment of the cocaine-exposed infant has a significant impact on regulatory problems during later infancy. Specifically, mothers who use heavier amounts of cocaine have higher levels of anxiety which, in turn, impact the sleep behaviors of their infants beyond the first month of life. Thus, interventions which include services to reduce maternal anxiety may have a positive impact on infant regulation. Another explanation for these findings is that infants who were prenatally exposed to heavier amounts of cocaine display disrupted regulatory processes beginning at or immediately after birth, which then increases maternal anxiety. In the latter scenario, the increased sleep difficulties seen in heavily exposed 7-month-old infants demonstrate continuity in disrupted behavioral-state organization from the neonatal period. This explanation is supported by our findings that sleep behaviors at 7 months of age were associated with increased physiological arousal and dysregulation during periods of rest at both 1 and 7 months.

Importantly, there was no association between maternal cocaine use during pregnancy and maternal cognitions about their infants' sleep behaviors. Although there was a slight association between cocaine use during pregnancy and scores on the Doubt subscale of the MCISQ, this difference disappeared after controlling for maternal alcohol consumption. Thus, mothers who used cocaine during pregnancy do not appear to think differently about the sleep behaviors of their infants than do mothers who did not use cocaine during pregnancy. Thus, although parental cognitive factors have been associated with the development of various behavior problems in children (see Morrell, 1999b), these findings suggest that maternal cognitions about sleep are unrelated to the sleep difficulties found among heavily exposed infants.

Within the cocaine group, there were significant differences in perinatal risk between infants who remained in parental care at 7 months of age and those who did not. Specifically, the infants in nonparental care were exposed to heavier amounts of cocaine, had decreased fetal growth, fewer prenatal visits, and less optimal scores on the OCS; however, despite their higher perinatal risk, infants who were in nonparental care at 7 months had less severe sleep problems than did infants who remained in parental care. Since the nonmaternal caregivers had significantly fewer symptoms of psychopathology than the cocaine-using women who retained custody of their children, one possible explanation for this finding is that the quality of the caregiving environment has a significant impact on infant sleep. Another possible explanation for these findings is that the mothers who retained custody of their children were more likely to perceive sleep problems among their infants than were other caregivers. In fact, there has been considerable discussion regarding the possible influence of maternal psychiatric symptoms such as depression on maternal reports of children's behavior (e.g., Briggs-Gowan, Carter, & Schwab-Stone, 1996; Chilcoat & Breslau, 1997; Fergusson, Lynskey, & Horwood, 1993). Sophisticated analytic models have demonstrated support for both the hypothesis that children of mothers with psychiatric problems have higher behavior problems and the hypothesis that mothers with a history of psychiatric disorder overreport negative behavior in their children (Chilcoat & Breslau, 1997). Thus, it is possible that higher sleep problems noted among the cocaine-exposed infants in the care of their biological mothers were due to the higher rates of maternal psychiatric symptoms in this group, method bias, or both; however, our findings of an association between sleep problems at 7 months of age and physiological regulation during sleep at 4 to 8 weeks of age suggest that at least in early infancy, exposed infants do have autonomic nervous system

functioning that differs from comparison infants. It is therefore likely that exposed infants exhibit differential physiological regulation early in life, which in turn contributes to maternal perceptions of more difficult behaviors later in infancy. Future studies are needed to examine whether the differential physiological regulation seen at 4 to 8 weeks of age predicts continued physiological dysregulation later in infancy and whether physiological regulation at 7 months of age is associated with maternal reports of infant sleep.

It is not surprising, then, that maternal psychopathology was so strongly related to maternal cognitions about their infants' sleep. Higher levels of caregiver psychopathology were associated with higher levels of maternal anger related to the demands of an infant, doubt about the adequacy of their parenting, beliefs that feeding is important for soothing an infant at night, and concerns about SIDS. Since maternal cognitions about infant sleep are associated with infant sleep problems (Morrell, 1999b), treatment for psychiatric symptoms may ameliorate the development of sleep problems.

There are other limitations of this study. First, the number of infants exposed to heavier amounts of cocaine as well as the number of cocaine-exposed infants in nonparental care were relatively small, increasing the chance that a few extreme data points may have influenced the findings. Second, it is unclear if the finding of sleep problems among cocaine-exposed infants, particularly those who remain in parental care, at 7 months of age is a transient problem that may lessen as a result of normal developmental processes. Longitudinal studies have shown that sleeping problems tend to decline with age (Kataria, Swanson, & Trevarthan, 1987). Thus, future studies should use a longitudinal approach to examining sleep behaviors in drug-exposed infants to determine if the increased sleep problems among cocaine-exposed infants at 7 months of age is a transient issue or whether they predict a more persistent problem. This is particularly important given the finding by some studies that persistent sleep problems are associated with psychological disturbances in early childhood (Herzog, 1980; Lozoff et al., 1985; Mahler et al., 1975), including both internalizing and externalizing problems (Atkinson, Vetere, & Grayson, 1995; Seifer et al., 1996). Third, our measures of sleep behaviors are based on maternal report, which is susceptible to bias, particularly among women with increased psychopathological symptomatology; however, our objective findings of dysregulated physiological behavior during sleep and rest among infants reported to have more sleep problems suggest that these findings reflect the actual presence of sleep disturbances among cocaine-exposed infants rather than just altered maternal perceptions. Thus, future studies should include objective measures of sleep when exploring the development of these behaviors among substance-exposed infants. Finally, although care was taken in the present study to identify substance use in this sample, the accurate assessment of substance use is difficult. Pregnant women are often hesitant to divulge information regarding the use of substances during pregnancy, particularly illicit substances such as cocaine. To address this issue, multiple indices of cocaine use were used, including self-report using the reliable Timeline Followback Interview as well as analysis of hair and urine samples. Each of these measures has its own limitations; however, when used in combination, the likelihood of accurately identifying cocaine use is increased. Note that measures of the other substance use during pregnancy were based entirely on self-report.

Despite these limitations, the present findings are important because they provide additional support for the influence of prenatal exposure to cocaine on infant regulation and indicate that one pathway from cocaine exposure to altered regulation in older infants may be through the caregiving environment. In particular, these findings highlight the importance of targeting psychiatric symptoms in addition to substance use in clinical interventions. In addition, this study adds to an increasing literature suggesting that cocaine-exposed infants who remain in parental care experience developmental outcomes that differ from those of

infants in nonparental care. Finally, similar to the findings of other studies (Jacobson et al., 1996; Lester et al., 2003), our findings suggest that altered developmental outcomes become apparent at heavier levels of exposure and highlight the importance of considering a dose-dependent response when examining the development of cocaine-exposed children.

Acknowledgments

The authors thank the parents and infants who participated in this study and the research staff who were responsible for conducting numerous assessments with these families. Special thanks to Drs. Claire Coles and Philip S. Zeskind for their collaboration on this study, to Drs. Amol Lele and Luther Robinson for collaboration on data collection at the Women and Children's Hospital of Buffalo, and to Dr. Michael Ray for his collaboration on data collection at Sisters of Charity Hospital of Buffalo. This study was made possible by NIDA Grant 1R01DA013190–01A2.

References

Adair R, Bauchner H, Philipp B, Levenson S, Zuckerman B. Night waking during infancy: Role of parental presence at bedtime. Pediatrics. 1991; 87:500–504. [PubMed: 2011427]

Anders TF. Infant sleep, nighttime relationships, and attachment. Psychiatry. 1994; 57:11–21. [PubMed: 8190824]

Anders TF, Keener M. Developmental course of nighttime sleep–wake patterns in full-term and premature infants during the first year of life. Vol. 1. Sleep. 1985; 8:173–192. [PubMed: 4048734]

Atkinson E, Vetere A, Grayson K. Sleep disruption in young children: The influence of temperament on the sleep patterns of pre-school children. Child Care, Health and Development. 1995; 21:233–246.

Baron RM, Kenny DA. The moderator-mediatory variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. Journal of Personality and Social Psychology. 1986; 51:1173–1182. [PubMed: 3806354]

Baumgartner WA, Hill VA, Blahd WH. Hair analysis for drugs of abuse. Journal of Forensic Science. 1989; 34:1433–1453.

Beauchaine TP. Vagal tone, development and Gray's motivational theory: Toward an integrated model of autonomic nervous system functioning in psychopathology. Development and Psychopathology. 2002; 13:183–214. [PubMed: 11393643]

Beckwith L, Howard J, Espinosa M, Tyler R. Psychopathology, mother–child interaction, and infant development: Substance-abusing mothers and their offspring. Development and Psychopathology. 1999; 11:715–725. [PubMed: 10624722]

Bendersky M, Lewis M. Prenatal cocaine exposure and impulse control at 2 years. Annals of the New York Academy of Sciences. 1998a; 846:365–367. [PubMed: 9668426]

Bendersky M, Lewis M. Arousal modulation in cocaine-exposed infants. Developmental Psychology. 1998b; 34:555–564. [PubMed: 9597364]

Benoit D, Zeahnah CH, Boucher C, Minde KK. Sleep disorders in early childhood: Association with insecure maternal attachment. Journal of the American Academy of Child and Adolescent Psychiatry. 1992; 31:86–93. [PubMed: 1537786]

Bernal JF. Night waking in infants during the first 14 months. Developmental Medicine and Child Neurology. 1973; 15:760–769. [PubMed: 4767650]

Blurton Jones N, Rosseti Ferreira MC, Farguhar Brown M, MacDonald L. The association between perinatal factors and late night waking. Developmental Medicine and Child Neurology. 1978; 20:427–434. [PubMed: 689308]

Bornstein, MH.; Lamb, ME., editors. Developmental psychology: An advanced textbook. Hillsdale, NJ: Erlbaum; 1992.

Boulet J, Boss MW. Reliability and validity of the Brief Symptom Inventory. Psychological Assessment. 1991; 3:433–437.

Boyd CJ. The antecedents of women's crack cocaine use: Family substance abuse, sexual abuse, depression, and illicit drug use. Journal of Substance Abuse Treatment. 1993; 10:433–438. [PubMed: 8246316]

Brandon TH, Copeland AL, Saper ZL. Programmed therapeutic messages as a smoking treatment adjunct: Reducing the impact of negative affect. Health Psychology. 1995; 14:41–47. [PubMed: 7737072]

Brown J, Bakeman R, Coles C. Maternal drug use during pregnancy: Are preterms and full-terms affected differently? Developmental Psychology. 1998; 34:540–554. [PubMed: 9597363]

Brown JV, Bakeman R, Coles CD, Platzman KA, Lynch ME. Prenatal cocaine exposure: A comparison of 2-year-old children in parental and nonparental care. Child Development. 2004; 75:1282–1295. [PubMed: 15260878]

Buena Vista Home Entertainment. Discover Spot [video tape]. 2000

Calkins SD. Cardiac vagal tone indices of temperamental reactivity and behavioral regulation in young children. Developmental Psychobiology. 1997; 31:125–135. [PubMed: 9298638]

Chilcoat HD, Breslau N. Does psychiatric history bias mothers' reports? An application of a new analytic approach. Journal of the American Academy of Child and Adolescent Psychiatry. 1997; 36:971–979. [PubMed: 9204676]

Chavin W, Tinson S. Children with sleep difficulties. Health Visitor. 1980; 53:477–481. [PubMed: 6907217]

Dennis C, Lori B. Relationships among infant sleep patterns, maternal fatigue, and development of depressive symptomatology. Issues in Perinatal Care. 2005; 32:187–193.

Derogatis, LR. The Brief Symptom Inventory (BSI) administration, scoring, and procedures manual. Minneapolis, MN: National Computer Systems; 1993.

Dickstein S, Seifer R, Hayden LC, Schiller M, Sameroff AJ, Keitner G, Miller I, Rasmussen S, Matzko M, Magee KD. Levels of family assessment: II. Impact of maternal psychopathology on family functioning. Journal of Family Psychology. 1998; 12:23–40.

Dinges DF, Davis MM, Glass P. Fetal exposure to narcotics: Neonatal sleep as a measure of nervous system disturbance. Science. 1980; 209:619–621. [PubMed: 7190326]

DiPietro JA, Caughy MO, Cusson RC, Fox NA. Cardiorespiratory functioning of preterm infants: Stability and risk associations for measures of heart rate variability and oxygen saturation. Developmental Psychobiology. 1994; 27:137–152. [PubMed: 8200487]

DiPietro JA, Suess PE, Wheeler JS, Smouse PhH, Newlin DB. Reactivity and regulation in cocaine-exposed infants. Infant Behavior and Development. 1995; 18:407–414.

Eiden RD, Lewis A, Croff S, Young E. Maternal cocaine use and infant behavior. Infancy. 2002; 3:77–96.

Eiden RD, Peterson M, Coleman T. Maternal substance use and the caregiving environment during early childhood. Psychology of Addictive Behaviors. 1999; 13:293–302.

Fergusson DM, Lynskey MT, Horwood LJ. The effect of maternal depression on maternal ratings of child behavior. Journal of Abnormal Child Psychology. 1993; 21:245–269. [PubMed: 8335763]

Field TM. Interventions in early infancy. Infant Mental Health Journal. 1992; 13:329–336.

Gawin FH, Ellinwood EH. Cocaine and other stimulants: Actions, abuse, and treatment. New England Journal of Medicine. 1988; 318:1173–1182. [PubMed: 3283549]

Gelfand DM, Teti DM. The effects of maternal depression on children. Clinical Psychology Review. 1990; 10:329–353.

Gingras JL, Feibel JB, Dalley LB, Muelenaer A, Knight CG. Maternal polydrug use including cocaine and postnatal sleep architecture: Preliminary observations and implications for respiratory control and behavior. Early Human Development. 1995; 43:197–204. [PubMed: 8835189]

Gingras JL, Weese-Mayer DE, Hume RF Jr, O'Donnell KJ. Cocaine and development: Mechanisms of fetal toxicity and neonatal consequences. Early Human Development. 1992; 31:1–24. [PubMed: 1486815]

Grossman D. Respiration, stress and cardiovascular function. Psychophysiology. 1983; 20:284–299. [PubMed: 6408680]

Halpern LF, Anders TF, Garcia Coll C, Hua J. Infant temperament: Is there a relation to sleep–wake states and maternal nighttime behavior? Infant Behavior and Development. 1994; 17:255–263.

Hans SL. Studies of prenatal exposure to drugs: Focusing on parental care of children. Neurotoxicology & Teratology. 2002; 24:329–337. [PubMed: 12009488]

Herzog JM. Sleep disturbance and father hunger in 18- to 28-month-old boys: The Erlkonig syndrome. Psychoanlaytic Study of the Child. 1980; 35:219–233.

Hume RF, O'Donnell KJ, Stanger CL, Killam AP, Gingras JL. In utero cocaine exposure: Observations of fetal behavioral state may predict neonatal outcome. American Journal of Obstetrics & Gynecology. 1989; 161:685–690. [PubMed: 2675600]

IBI Analysis Software 7.485. James Long Company; Caroga Lake, NY: 1999.

Jacobson SW, Jacobson JL, Sokol RJ, Martier SS, Chiodo LM. New evidence for neurobehavioral effects of in utero cocaine exposure. Journal of Pediatrics. 1996; 129:581–590. [PubMed: 8859266]

Judd CM, Kenny DA. Process analysis: Estimating mediation in treatment evaluations. Evaluation Review. 1981; 5:602–619.

Karmel BZ, Gardner JM. Prenatal cocaine exposure effects on arousal modulated attention during the neonatal period. Developmental Psychobiology. 1996; 29:463–480. [PubMed: 8809496]

Kataria S, Swanson MS, Trevarthan GE. Persistence of sleep disturbances in preschool children. Journal of Pediatrics. 1987; 110:642–646. [PubMed: 3559818]

Kettinger LA, Nair P, Schuler M. Exposure to environmental risk factors and parenting attitudes among substance-abusing women. American Journal of Drug & Alcohol Abuse. 2000; 26:1–11. [PubMed: 10718159]

Lester BM, LaGasse LL, Seifer R. Cocaine exposure and children: The meaning of subtle effects. Science. 2003; 282:633–634. [PubMed: 9841414]

Littman A, Parmelee B. Medical correlation of infant development. Pediatrics. 1978; 61:474.

Lozoff B, Wolf AW, Davis NS. Sleep problems seen in pediatric practice. Pediatrics. 1985; 75:477–483. [PubMed: 3975116]

Luthar SS, Cushing G, Merikangas KR, Rounsaville BJ. Multiple jeopardy: Risk and protective factors among addicted mothers' offspring. Development and Psychopathology. 1998; 10:117–136. [PubMed: 9524811]

MacKinnon, DP. Analysis of mediating variables in prevention and intervention research. In: Cazares, A.; Beatty, LA., editors. Scientific methods in prevention research. NIDA research monograph. Vol. 139. Washington, DC: U.S. Govt. Print Office; 1994. p. 127-153.DHHS Pub. No. 94–3631

MacKinnon DP, Krull JL, Lockwood CM. Equivalence of the mediation, confounding, and suppression effect. Prevention Science. 2000; 1:173–181. [PubMed: 11523746]

MacKinnon DP, Lockwood CM, Hoffman JM, West SG, Sheets V. A comparison of methods to test mediation and other intervening variable effects. Psychological Methods. 2002; 7:83–104. [PubMed: 11928892]

Magura S, Freeman RC, Siddiqi Q, Lipton DS. The validity of hair analysis for detecting cocaine and heroin use among addicts. Journal of the Addictions. 1992; 27:51–69.

Mahler, M.; Pine, F.; Bergman, A. The psychological birth of the human infant. New York: Basic Books; 1975.

Mayes LC, Bornstein MH, Chawarska K, Granger RH. Information processing and developmental assessments in 3-month-old infants exposed prenatally to cocaine. Pediatrics. 1995; 95:539–545. [PubMed: 7700755]

Mayes LC, Bornstein MH, Chawarska K, Granger RH. Impaired regulation of arousal in three-month-old infants exposed to cocaine and other drugs. Developmental Psychopathology. 1996; 8:29–42.

Moore T, Ucko LE. Night waking in early infancy: Part 1. Archives of Diseases in Childhood. 1957; 32:333–342. [PubMed: 13459463]

Morrell JMB. The infant sleep questionnaire: A new tool to assess infant sleep problems for clinical and research purposes. Child Psychology and Psychiatry Review. 1999a; 4:20–26.

Morrell JMB. The role of maternal cognitions in infant sleep problems as assessed by a new instrument, the Maternal Cognitions About Infant Sleep Questionnaire. Journal of Child Psychology & Psychiatry. 1999b; 40:247–258. [PubMed: 10188707]

Nassogne, MC.; Evrard, P.; Courtoy, PJ. Selective direct toxicity of cocaine on fetal mouse neurons. Teratogenic implications of neurite and apoptotic neuronal loss. In: Harvey, JA.; Kosofsky, BE.,

editors. Cocaine: effects on the developing brain. Annals of the New York Academy of Sciences. Vol. 846. New York: New York Academy of Sciences; 1998. p. 51-68.

Paret, I. Night waking and its relation to mother–infant interaction in nine-month-old infants. In: Call, JD.; Galenson, E., editors. Frontiers of infant psychiatry. Vol. 1. New York: Basic Books; 1983. p. 171-177.

Regalado M, Schechtman V, Del Angel AP, Bean X. Sleep disorganization in cocaine-exposed neonates. Infant Behavior and Development. 1995; 18:319–327.

Regalado M, Schechtman V, Del Angel AP, Bean X. Cardiac and respiratory patterns during sleep in cocaine-exposed neonates. Early Human Development. 1996; 44:187–200. [PubMed: 8654312]

Richman N. A community survey of characteristics of one- to two-year-olds with sleep disruptions. Journal of the American Academy of Child Psychiatry. 1981; 20:281–291. [PubMed: 7264107]

Richman N, Douglas J, Heather H, Lansdown R, Levere R. Behavioral methods in the treatment of sleep disorders—a pilot study. Child Psychology and Psychiatry. 1985; 26:581–590.

Robbins TW. Arousal systems and attentional processes. Biological Psychology. 1997; 45:57–71. [PubMed: 9083644]

Sameroff, AJ.; Chandler, M. Reproductive risk and the continuum of caretaking casualty. In: Horowitz, FD., editor. Review of child development research. Vol. 4. Chicago: University of Chicago Press; 1975. p. 187-244.

Scher MS, Richardson GA, Coble PA, Day NL, Stoffer DS. The effects of prenatal alcohol and marijuana exposure: Disturbances in neonatal sleep cycling and arousal. Pediatric Research. 1988; 24:101–105. [PubMed: 3412843]

Scher MS, Richardson GA, Day NL. Effects of prenatal cocaine/crack and other drug exposure on electroencephalographic sleep studies at birth and one year. Pediatrics. 2000; 105:39–48. [PubMed: 10617702]

Schuetze P, Eiden RD. The association between maternal cocaine use during pregnancy and physiological regulation in four- to eight-week-old infants: An examination of possible mediators and moderators. Journal of Pediatric Psychology. 2006; 31:15–26. [PubMed: 15788714]

Schuetze P, Eiden RD. The association between maternal smoking and secondhand exposure and autonomic functioning at 2–4 weeks of age. Infant Behavior and Development. 2006; 29:32–43. [PubMed: 17138259]

Seifer R, Sameroff AJ, Dickstein S, Hayden L, Schiller M. Parental psychopathology and sleep variation in children. Child and Adolescent Psychiatric Clinics of North America. 1996; 5:715–727.

Singer LT, Arendt R, Farkas K, Minnes S, Huang J, Yamashita T. The relationship of prenatal cocaine exposure and maternal psychological distress to child development. Development and Psychopathology. 1997; 9:473–489. [PubMed: 9327234]

Singer L, Salvator A, Arendt R. Effects of cocaine/polydrug exposure and maternal psychological distress on infant birth outcomes. Neurotoxicology and Teratology. 2002; 24:127–135. [PubMed: 11943500]

Sobell, ME. Asymptotic confidence intervals for indirect effects in structural equation models. In: Leinhardt, S., editor. Sociological methodology 1982. Washington, DC: American Sociological Association; 1982. p. 290-312.

Sobell ME, Sobell LC, Klajner F, Pavan D, Basian E. The reliability of a timeline method for assessing normal drinker college students' recent drinking history. Addictive Behaviors. 1986; 11:149–162. [PubMed: 3739800]

Tucker DM, Williamson PA. Asymmetric neural control systems in human self-regulation. Developmental Psychology. 1984; 91:185–215.

Wasserman DR, Leventhal JM. Maltreatment of children born to cocaine-dependent mothers. American Journal of Diseases of Children. 1993; 147:1324–1328. [PubMed: 8249955]

Weissbluth M. Brief clinical and laboratory observations: Sleep duration and infant temperament. Journal of Pediatrics. 1981; 99:817–819. [PubMed: 7299565]

Zuckerman B, Stevenson J, Bailey V. Sleep problems in early childhood: Continuities, predictive factors, and behavioral correlates. Pediatrics. 1987; 80:664–671. [PubMed: 3670967]

Infant Ment Health J. Author manuscript; available in PMC 2011 July 18.

TABLE 1

Group Differences for Level of Cocaine Exposure

	Abstainers (*n* = 53)		Light Users (*n* = 39)		Heavy Users (*n* = 26)		
	M	***SD***	***M***	***SD***	***M***	***SD***	***F***
Maternal Characteristics							
Age (years)	29.83[a]	5.41	32.44[b]	4.64	31.54	5.72	2.98*
Parity	3.32[a]	1.69	4.69[b]	2.27	3.8	1.85	5.67**
Parental care (No. of visits)	15.02[a]	8.24	13.3	8.68	8.53[b]	7.2	3.97*
No. of days cocaine used during pregnancy	0[a]	0	7.77[a]	15.13	109.08[b]	67.79	106.16**
No. of cigarettes smoked during pregnancy	460.38[a]	985.79	1080.13	1093.21	1920.54[b]	1948.60	11.36**
No. of standard drinks	6.25[a]	28.08	83.74[a]	235.89	479.31[b]	822.44	12.25**
No. of binge-drinking episodes	.94[a]	4.18	3.10[a]	8.77	47.73[b]	83.77	13.72**
No. of joints smoked	41.68	272.11	26.56	76.73	65.58	143.92	.30
BSI Subscales							
Somaticism	2.4	3.5	2.77	3.63	3.35	4.71	.54
Phobic Anxiety	.17[a]	.38	.45	.71	.68[b]	.68	7.2**
Anxiety	.27[a]	.36	.68[b]	.88	.82[b]	.99	6.4**
Obsessive Compulsive	.84	.81	.80	.91	.71	.90	.21
Interpersonal Sensitivity	.44	.56	.63	.87	.54	.91	.72
Depression	.41	.58	.48	.79	.40	.72	.15
Hostility	.58	.60	.53	.70	.55	.83	.06
Paranoid Ideation	.83	.75	1.01	1.08	.82	1.03	.51
Psychoticism	.42	.52	.52	.65	.50	.75	.33
Infant Characteristics							
Gestational age	39.3[a]	1.4	38.6	1.9	38.0[b]	2.2	5.39**
Birth weight (g)	3406.49[a]	573.15	3057.33[b]	554.54	2678.38[c]	454.19	16.20**
Birth length (cm)	49.69[a]	3.29	48.83	2.71	47.46[b]	2.27	4.85**
Head circumference at birth (cm)	33.67	1.43	33.2	1.27	32.93	1.34	2.88
OCS	92.02[a]	12.91	83.41[b]	13.84	80.75[b]	14.78	7.38**

	Abstainers (*n* = 53)		Light Users (*n* = 39)		Heavy Users (*n* = 26)		
	M	***SD***	***M***	***SD***	***M***	***SD***	***F***
ISQ	3.8[a]	4.7	2.59[a]	1.11	6.26[b]	5.72	5.67**
MCISQ total	1.69	1.08	1.88	1.19	1.52	.64	.97
Setting limits	2.94	1.31	3.09	.89	3.39	1.39	1.44
Anger	.91	1.21	.89	1.23	1.1	1.47	1.44
Doubt	1.25[a]	1.21	1.35[a]	1.22	1.63[b]	1.41	2.96*
Feeding	1.51	1.51	1.50	1.39	1.86	1.60	.79
Safety	1.13	1.47	1.27	1.43	1.27	1.44	.15

Numbers that do not share the same superscript (“a” or “b”) are significantly different from one another at $p < .05$ levels; numbers that share the same superscript are not significantly different. BSI=Brief Symptom Inventory; OCS=obstetrical Complications Scale; ISQ=Infant Sleep Questionnaire; MSISQ=Maternal Cognitions About Infant Sleep Questionnaire.

* $p < .05$.

** $p < .01$.

TABLE 2

Correlations Among Substance Use During Pregnancy, Psychopathology and Sleep Variables

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.	21.
1. Cocaine	–																				
2. Marijuana-No. of joints	−.00	–																			
3. No. of Standard drinks	.57**	.00	–																		
4. Binge episodes	.59**	−.02	−.94**	–																	
5. No. of cigarettes	.40**	.00	.34**	.39**	–																
6. BSI-Somaticism	.08	−.03	.12	.11	.16	–															
7. BSI-Obsessive complusive	.01	−.06	.17	.17	.12	.65**	–														
8. BSI-Interpersonal sensitivity	.04	−.05	.22*	.21*	.14	.51**	.64**	–													
9. BSI-Depression	.003	.02	.17	.13	.03	.64**	.71**	.78**	–												
10. BSI-Anxiety	.19*	−.00	.24**	.24**	.08	.63**	.66**	.66**	.78**	–											
11. BSI-Hostility	.05	−.01	.30**	.26**	.13	.41**	.54**	.63**	.62**	.53**	–										
12. BSI-Phobic anxiety	.18*	−.07	.23**	.21*	.11	.52**	.50**	.46**	.55**	.63**	.39**	–									
13. BSI-Paranoid ideation	−.02	−.03	.18*	.14	.12	.58**	.60**	.79**	.70**	.66**	.67**	.59*	–								
14. BSI-Psychoticism	.07	−.15	.13	.13	.15	.57**	.61**	.73**	.78**	.69**	.55**	.56*	.71**	–							
15. ISQ Total	−.11	−.09	.03	−.01	.02	.06	−.01	.04	−.02	.18*	.00	.19*	.00	−.05	–						
16. MCISQ-Total	−.03	−.10	.12	.11	−.04	.11	.13	.20*	.09	.10	.30**	.19*	.32**	.13	.16	–					
17. MCISQ–Setting limits	−.00	−.02	.02	.05	−.09	.02	−.04	.01	−.04	−.06	.05	.09	.12	.00	.09	.80**	–				
18. MCISQ-Anger	−.05	−.08	.09	.05	−.02	.16	.20*	.23*	.25**	.19*	.41**	.23**	.33**	.23*	.17	.84**	.50**	–			
19. MCISQ-Doubt	−.11	−.06	.08	.07	−.02	.12	.11	.20*	.09	.10	.29**	.19**	.35**	.12	.12	.90**	.63**	.76**	–		
20. MCISQ-Feeding	.05	−.13	.24**	.24**	−.06	.05	.06	.14	−.05	−.06	.16	.07	.19*	.01	.10	.72**	.46**	.46**	.56**	–	
21.MCISQ-Safety	.05	−.14	.06	.07	.06	.07	.11	.25**	.06	.10	.32**	.15	.30**	.17	.13	.63**	.49**	.49**	.57**	.37**	–

BSI=Brief Symptom Inventory; ISQ=Infant Sleep Questionnaire; MCISQ=Maternal Cognitions About Infant Sleep Questionnaire.

*$p < .05$.

**$p < .01$.

Infant Ment Health J. Author manuscript; available in PMC 2011 July 18.

TABLE 3

Group Differences by Caregiving Status

	Parental		Nonparental Custody		Nonexposed		
	M	*SD*	*M*	*SD*	*M*	*SD*	*F*
Maternal Characteristics							
Age (years)	33.02[a]	4.05	29.61[b]	6.62	29.69[b]	5.37	6.7**
Parity	4.42[a]	2.22	4.12[a]	1.96	3.35[b]	1.7	4.25*
Prental care (No. of visits)	8.65[a]	15.02	5.89[a]	4.11	15.02[b]	8.24	3.87*
No. of days cocaine used during pregnancy	35.23[a]	63.88	82.39[b]	62.8	0[c]	0	15.74**
No. of cigarettes smoked during pregnancy	1391.3	1633.21	1481.56	1296.01	469.23	993.28	2.51
No. of standard drinks	314.94[a]	665.99	51.5	96.79	6.36[b]	28.34	8.98**
No. of joints smoked	26.98	71.53	81.83	169.91	42.48	274.70	.50
BSI Subscales							
Somaticism	3.47	4.26	1.78	3.32	2.38	3.53	1.66
Phobic Anxiety	.47[a]	.73	.18[b]	.45	.16[b]	.39	5.93**
Anxiety	.73[a]	1.02	.12[b]	.25	.35[b]	.44	7.77**
Obsessive Compulsive	.94[a]	.96	.31[b]	.50	.84[a]	.82	3.87*
Interpersonal Sensitivity	.77[a]	.97	.13[b]	.21	.44[c]	.56	5.85**
Depression	.59[a]	.83	.07[b]	.28	.41[a]	.58	4.42*
Hostility	.69	.82	.14	.28	.58	.60	.71
Paranoid Ideation	1.16[a]	1.14	.33[b]	.41	.82[a]	.76	5.76**
Psychoticism	.61	.74	.26	.43	.42	.52	2.54
Infant Characteristics							
Gestational Age	38.75[a]	1.74	37.94[b]	2.25	39.31[a]	1.37	5.21**
Birthweight (grams)	3053.1[a]	535.3	2577.2[b]	405.9	3423.5[c]	565.0	10.87**
Birthlength (cm)	48.8[a]	2.47	46.96[b]	2.57	49.7[a]	3.32	3.75*
Head Circumference at birth (cm)	33.21	1.29	32.79	1.28	33.7	1.42	1.34
OCS	82.55[a]	13.65	81.94[a]	15.99	92.33[b]	12.84	7.4**

	Parental		Nonparental Custody		Nonexposed		
	M	***SD***	***M***	***SD***	***M***	***SD***	***F***
ISQ	5.81[a]	5.32	2.15[b]	.69	3.8[b]	4.7	4.74**
MCISQ total	1.85	1.11	1.44	.63	1.69	1.08	1.01
Setting limits	3.36	1.33	3.01	.84	2.94	1.31	1.5
Anger	1.00	1.42	.58	.66	.91	1.21	.78
Doubt	1.61	1.29	1.4	.71	1.36	1.21	.59
Feeding	1.62	1.49	1.22	1.35	1.86	1.60	1.23
Safety	1.36	1.47	1.03	1.31	1.12	1.47	.50

Numbers that do not share the same superscript (“a” or “b”) are significantly different from one another at $p < .05$ levels; numbers that share the same superscript are not significantly different. BSI=Brief Symptom Inventory; OCS=obstetrical Complications Scale; ISQ=Infant Sleep Questionnaire; MSISQ=Maternal Cognitions About Infant Sleep Questionnaire.

Note:

* $p < .05$.

** $p < .01$.

TABLE 4

Correlations Among Sleep and Physiological Regulation Variables

		1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1.	ISQ Total	–										
2.	MCISQ Total	.16	–									
3.	MCSIQ-Setting Limits	.09	.80***	–								
4.	MCISQ-Anger	.17	.84***	.50***	–							
5.	MCISQ-Doubt	.12	.90***	.63***	.76***	–						
6.	MCISQ-Feeding	.10	.72***	.46***	.46***	.56***	–					
7.	MCISQ-Safety	.13	.63***	.49***	.49***	.57***	.37**	–				
8.	HR at 4–8 weeks	.04	.35**	.29**	.21*	.42***	.25*	.25*	–			
9.	RSA at 4–8 weeks	−.31**	−.20*	−.20*	−.24*	−.21*	−.11	−.14	−.12	–		
10.	HR at 7 months	.05	.03	.01	.01	−.08	−.04	.01	.20	.13	–	
11.	RSA at 7 months	−.27*	−.57***	−.36**	−.55***	−.53***	−.48**	−.47**	−.02	.22*	−.22*	–*

ISQ= Infant sleep Questionnaire, MCSIQ=Maternal Cognitions About Infant Sleep Questionnaire; HR= heart rate; RSA= respiratory sinus arrhythmic. $p < .05$.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

TABLE 5

Hierarchical Linear Regression Models—Mediational Analyses for Infant Sleep Questionnaire (ISQ)

Predictor Variables	Unstandardized coefficients β	SE	Standardized β	R^2	F
Regression 1: Outcome—Anxiety					
Abstainers vs. users	−.13	.18	−.07	.32	6.4**
Abstainers/light vs. heavy users	−.42	.15	−.28**		
Regression 2: Outcome—ISQ					
Abstainers vs. users	3.31	1.21	.28**	.07	4.16*
Abstainers/light vs. heavy users	−2.1	1.01	−.22**		

* $p < .05$.

** $p < .01$.