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Sleep Architecture in Infants of Substance Abusing Mothers

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Abstract

Sleep Architecture in Infants of Substance Abusing Mothers

This longitudinal, year-long study compared sleep-wake state organization in two groups of infants: infants whose mothers abused substances during their pregnancies, and non-exposed, typically developing, age-matched comparison infants, to determine whether differences in sleep-wake state organization existed between the two groups. Seventeen infants of mothers who were participating in a parent-infant residential treatment program for substance abuse were enrolled. Their sleep-wake state organization over the first year of life was compared to that of 17 age-matched comparison infants. The intent was to follow each infant on 5 occasions over the first year of life using established methods of time-lapse videosomnography to record sleep-wake state organization. However, attrition in the substance-abusing group was problematic.

Some sleep-wake variables (Active Sleep%, Quiet Sleep%, Awake%, number of nighttime awakenings) were similar for both groups of infants at comparable ages across the first year. Total sleep time and the longest sustained sleep period (sleep continuity variables) differed significantly at some of the ages measured. Although overall sleep architecture appears highly resilient and well organized, some indications of sleep fragmentation and shortened nighttime sleep periods were observed in the substance-exposed infants. More research is needed to explain why sleep continuity variables and not sleep state proportion variables differed between the two groups.

Sleep Architecture in Infants of Substance Abusing Mothers

Use of substances by women during their pregnancy has been a public health concern for many years. In a nationally representative survey of drug use among pregnant woman by the National Institute of Drug Abuse (NIDA, 1996), 20.4 percent of respondents reported smoking cigarettes, 18.8 percent reported drinking alcohol, and 5.5 percent reported using illicit drugs, including cocaine, marijuana, methamphetamine, and others. Estimates may be low given that the data are self-reported. This translates minimally into hundreds of thousands of pregnant woman using substances each year, despite concerns that major developmental consequences for the fetus may be associated with substance abuse during gestation (Lester, Boukydis, & Twomey, 2000).

Although reports in the literature have been somewhat inconclusive, the illicit and licit use of substances during pregnancy has been associated with a number of potentially harmful effects (Dow-Edwards, 1993; Eisen et al., 1991). For example, cocaine is reported to be directly toxic to the developing fetus and to the normal physiology of the placenta. Disruption in placental/fetal vasculature has been reported (Church, 1993; Delaney-Black et al., 1996; Doberczak, Snanzer, Senie, & Kandall, 1988; Malanga & Kosofsky, 1999; Richardson & Day, 1991). Spontaneous abortion, decreased birth weight, birth length, and head circumference also have been associated with in-utero cocaine exposure (Chiriboga, Brust, Bateman, & Hauser, 1999; Coles, Platzman, Smith, James, & Falek, 1992). Abnormal EEGs, brainstem transmission times, cries and problems with autonomic stability have been reported in the neonate (Corwin, Lester, Sepkoski, McLaughlin, Kayne, & Golub, 1992; Doberczak et al., 1998; Scher, Richardson, & Day, 2000). Neurobehavioral processes such as habituation, attention, regulation of state, learning, language, play, and motor/tone also have been reported as negatively effected

by in-utero cocaine exposure. This includes decreases in social behavior and activity level (Chasnoff, 1993; Chiriboga, Brust, Bateman, & Hauser, 1999; Church, 1993; Coles et al., 1992; Corwin et al., 1992; Delaney-Black et al., 1996; Delaney-Black, Covington, Templin, Ager, Martier, & Sokol, 1998; Doberczak, 1998; Eisen et al., 1991; Malanga & Kosofsky, 1999; Mayes, Granger, Frank, Schottenfeld, & Bornstein, 1993; Richardson & Day, 1991; Schneider & Chasnoff, 1992; Stewart & Meeker, 1997; Tronick & Beeghly, 1999; Tronick, Frank, Cabral, Mirochnick, & Zuckerman, 1996; Volpe, 1992). Methamphetamine has been associated with dysmorphogenic effects such as clefting and cardiac anomalies, fetal death, reduced growth, and learning and behavioral problems (Stewart & Meeker, 1997). Alcohol and marijuana have been associated with complications of pregnancy that include impaired growth, language, intelligence, attentional and behavioral difficulties, and multiple other problems similar to those of infants exposed to cocaine in utero (Joffe & Chernick, 1990; Lester & Dreher, 1989; Streissguth, Barr, Sampson, Darby, & Martin, 1989; Zuckerman et al., 1989). The fetal alcohol syndrome has been well described, and is one of the leading causes of mental retardation. Although 20-25% of pregnant woman are estimated to use tobacco the effects of tobacco on the developing fetus remain controversial at least in part because of the mixture of tobacco and other substances. Nevertheless, many detrimental effects have been reported. Cigarette smoking during pregnancy has been associated with preterm birth, intrauterine growth retardation, increased perinatal morbidity and mortality; learning, attentional and behavioral difficulties through childhood, and adolescent conduct disorder (Brennan, Grekin, & Mednick, 1999; Milberger, Biederman, Faraone, Chen, & Jones, 1996; Orlebke, Knol, & Verhulst, 1997).

Direct effects of any of these substances in humans have been difficult to prove. This is at least in part due to the fact that substance-abusing populations have been notoriously difficult to

study. Along with problems of compliance and variable amounts of substance abuse that are very difficult to quantify, there are often many additional confounding factors, such as social and economic circumstances that include poverty, stress, trauma, physical and psychological abuse, and the use of more than one substance (Chasnoff, 1993; Chiriboga et al., 1999; Church, 1993; Coles et al., 1992; Johnson & Leff, 1999).

Various animal studies have provided a more controlled means of linking specific substances directly to biological consequences including CNS developmental disruption. There is evidence that drugs affect the formation of the fetal brain and its ability to function by acting on a molecular level during critical periods of development. In this way drugs may mimic the actions of physiologic neurotransmitters or somehow alter their function. By inducing alterations in different neurotransmitter systems, including their receptors and various signaling pathways, drugs affect cellular responses and gene expression. This consequently affects protein formation, growth factors, neuronal migration, growth, and cytoarchitecture (Malanga & Kosofsky, 1999). Thus, programs necessary for normal brain development, and ultimately CNS structure and function, are altered on behavioral, cognitive, and affective levels at least in animals.

Because substance abuse during pregnancy has been reported to disrupt fetal CNS development, and because the development of sleep and waking states (during gestation and postnatally) also reflects brain maturation, one might predict that in-utero substance exposure might disrupt normal sleep-wake pattern development. There have been only a few reports of infant sleep disturbances associated with maternal substance abuse during pregnancy. In 1979, Rosett and colleagues studied fourteen three-day-old infants whose mothers had been heavy drinkers throughout pregnancy, and compared them to a group of eight infants whose mothers reduced their heavy drinking to moderate drinking or abstained during the third trimester of

pregnancy and to a group of nine infants whose mothers were never heavy drinkers but abstained or drank moderately throughout pregnancy. The infants were studied for twenty-four hours using a bassinet sleep monitor. The infants of mothers who were heavy drinkers slept less than the second group of infants, and had a poorer quality of quiet sleep and more restlessness than the third group of infants. In 1988, Scher and colleagues reported that prenatal alcohol and marijuana exposure were associated with increased arousals, increased body movements, and decreased total sleep in neonates. In a polysomnographic study reported by Dahl (1998) of prenatal marijuana exposure, exposed children at three years of age were found to have a number of sleep disturbances including more arousals, greater awake time after falling asleep, increased body movements, and decreased sleep efficiency. In 1995, Ginghas and colleagues, in a study of neonates exposed to cocaine and other substances prenatally, reported the exposed group showed less sleep, more frequent arousals, and a higher proportion of active sleep. In 2000, Scher and colleagues, in a study of infants exposed to cocaine and other substances prenatally, reported on two-hour computer generated EEG sleep recordings on the second day of life and at one year postpartum. They found that infants with prenatal cocaine exposure demonstrated less well-developed spectral correlations at birth, and lower spectral EEG power at one year of age. In contrast, cocaine exposed and non-exposed infants did not differ in sleep architecture and phasic measures. However, prenatal alcohol, marijuana, and tobacco exposure was associated with increased levels of arousal. The extant literature thus provides some evidence that prenatal exposure to substances is associated with disrupted sleep.

The present study, using videosomnography, investigated sleep-wake state organization longitudinally over the first year of life in a group of infants with mothers who abused substances during their pregnancy and an age-matched typically developing comparison group of infants.

During the first year of life, as an infant matures, sleep consolidates so that the longest continuous sleep period lengthens and shifts to the nighttime, the proportion of active sleep decreases and the proportion of quiet sleep increases. Time out of the crib also decreases (Anders, Keener, Bowe, & Shoaff, 1982). It was predicted that these normal age differences of sleep-wake state variables would be disrupted in a group of infants exposed to substances in-utero.

Methods

Thirty-four infants, seventeen whose mothers were participating in a treatment program for new mothers who had used drugs during pregnancy, and seventeen community-based, non-exposed infants were compared in this study. All infants were full-term, with no major complications of pregnancy or delivery, and medically uneventful postpartum courses. The infants were matched for gender and age. All substance-abusing mothers used substances during at least part of their pregnancies. Cessation of substance abuse and onset of treatment varied across the seventeen mothers, with two mothers not beginning treatment until after the birth of their infants. Drugs used were alcohol, tobacco, cocaine, marijuana, and methamphetamine, alone and in various combinations. This information was elicited via self-report. Substance-abusing mothers were unemployed, living on public assistance, and in a drug rehabilitation program on entrance to the study. Their average age was twenty-eight years old (range = 20-38 years). Twenty-nine percent of the substance-abusing mothers were African-American, 41% were Caucasian, 18% were Hispanic, and 12% were unknown or mixed.

The comparison group was predominantly middle class and living in a university community. It was chosen from a larger sample of a study of normal infant sleep-wake state development; infants in the comparison group were matched to those in the substance-abusing

group based on gender and age at measurement. The mothers in the comparison group ranged in age from 23 to 37, with a mean age of thirty. There was no known substance abuse among the mothers of the comparison group. Seventy-six percent of comparison group mothers were Caucasian, 6% were Asian, 6% were Hispanic, and 12% were mixed or unknown.

The original design called for a year–long longitudinal study of both groups. Videotaping of nighttime sleep-wake organization was planned when the infants were one, three, six, nine, and twelve months of age. Two contiguous nights of video recordings were obtained at each age and scores for the two nights were averaged. In general, recording times for each of the contiguous nights commenced in the evening when each child was put into bed and ended in the morning when the child awakened. Bedtimes and wake up times were comparable between the two groups. However, inadequate compliance in the substance-abusing group of mothers resulted in attrition over the year. Three mothers in the substance-abusing group did not enter the study until their infants were three months of age. Eleven of the 17 participated in the videotaping at three months, nine at six months, five at nine months, and six at twelve months. Only two of the 17 substance-exposed infants were videotaped at all five ages. In contrast, there was no attrition in the control group.

The rationale for using time-lapse video recording in the home has been described previously, as have the methods of recording and scoring the videotapes for sleep and waking states (Anders & Keener, 1985; Anders, Keener, & Kraemer, 1985; Anders & Sostek, 1976). Briefly, a time-lapse video recorder (set at 12:1 time-lapse reduction) and a camera (with a low level illumination lens) were brought to the residential treatment center and/or home during the afternoon prior to recording. The camera was secured on a tripod that overlooked the crib and a microphone was placed nearby to record infant vocalizations. A time-code generator in the video

recorder continuously recorded clock time during the night. Mothers were asked to initiate the equipment when they put their infant into the crib for nighttime sleep and stop the recording when the child woke up for the day. Using this equipment, an entire night of sleep can be recorded onto one standard videotape. From the video recordings the percent of time in Active Sleep (AS%), Quiet Sleep (QS%), Wakefulness (AW%), and Out of Crib (OOC%), as well as the Longest Sleep Period (LSP), Total Sleep Time (TST), and Number of Awakenings (NAWK) were scored according to standard methods described previously (Anders & Keener, 1985; Anders, Keener & Kraemer, 1985; Anders & Sostek, 1976). All coders completed a blinded training, reliability process.

Statistical differences were computed using the Statistical Package for the Social Sciences (SPSS), version 10. Basic descriptive statistics were computed for each of the sleep variables. The control and substance-abusing groups were then compared using independent sample t-tests to detect potential differences between groups on each of the sleep variables. When the variances of the two samples were non-equivalent (as shown by the significance of Levine's test), the more conservative numbers assuming non-equal variances are reported.

Results

There were no significant differences between the substance-abusing and comparison groups in QS percent and AS percent at any age across the first year of life. Both groups showed the expected rise in QS percent and decline in AS percent from one to twelve months of age. In both groups it was not until twelve months of age that the proportion of QS exceeded fifty percent of the sleep time (Anders & Keener, 1985; Anders et al., 1985; Anders & Sostek, 1976; Anders & Taylor, 1994; Coons, 1987). Similarly, the percent of time awake was not significantly different between the two groups. Both groups showed a marked decrease in OOC

percent from the beginning to the end of the year. For both groups the major decrease in OOC percent occurred between months one and three. Although the actual number of nighttime awakenings appears to be different between the two groups, this difference was not statistically significant. The number of awakenings and the percent awake time seemed to follow similar developmental patterns over the year in both groups. Also, as was expected, in both groups the infants' sleep appeared to become consolidated as reflected in LSPs becoming longer in duration over the course of the year.

The only significant differences between the two groups were noted to be in the longest sleep period (LSP) and in total sleep time (TST) (Table 1). The LSP and TST times of the high-risk infants are visibly less than those of the comparison subjects at all ages, but especially in the second half of the year. However, the only statistically significant differences between the two groups occurred for LSP at six months (t = -2.66, $p < .05$) and TST at three, six, and twelve months (t = -2.92, -2.41, & -2.88, respectively, $p < .05$).

Discussion

Contrary to the initial prediction, the development of sleep and waking states over the first year of life in drug-exposed infants appeared remarkably similar to the pattern found in normally developing control infants. Sleep-wake state proportions in the two groups were similar across the first year of life, suggesting that intra-uterine exposure to drugs does not affect gross sleep-wake state organization. These results resemble those of other studies wherein the development of the sleep-wake states of conceptional age-matched prematurely born infants and full-term infants were comparable (Anders & Keener, 1985; Anders et al., 1985).

The significant differences between the two groups in LSP and TST, and the absence of significant differences in other sleep-wake variables, suggest that sleep system regulators must

be highly resilient and well organized. It may also suggest that fragmentation rather than sleep-wake state organization may be more reflective of intrauterine substance exposure, supporting other studies by Rosett et al (1979), Scher et al (1988), and Dahl (1998). The findings of this study are noteworthy, especially the integrity of sleep architecture, considering that offspring of substance abusing mothers are reported as being at risk for so many other problems and the association between prenatal drug exposure and neurodevelopmental vulnerability has been fairly well documented.

The absence of more widespread differences may be explained by a number of methodological reasons. Some of the trends that were visibly apparent but did not reach statistical significance may be nevertheless real but the sample size may have been too small to uncover true differences that may exist. In addition, although the sample of substance-abusing mothers and their infants was small, the much more relevant issue relates to attrition in this group compared to the comparison group. Because of this attrition, there were fewer infants to compare to the comparison group at the later ages of study and thus less power to detect differences that might actually exist between groups. In addition, it is possible that the infants with the most disturbed sleep-wake state organization are those that were lost to attrition. The lifestyle of the substance-abusing mothers was chaotic. Although great efforts were made to find participants at each scheduled measurement period, in a number of cases our attempts were unsuccessful despite the fact that measurement periods lapsed only 2 months.

Another concern is the inadequate matching of subjects. The subjects were matched only for gender and age because the socioeconomic status and lifestyle of the hi-risk mothers were too extreme to match to the comparison mothers. It is possible that the differences observed in time spent out of the crib in the middle of the night were due to documented SES and ethnic

differences in co-sleeping prevalence, and not substance exposure per se (e.g., Lozoff, Askew, & Wolf, 1996). Finally, the complicated interaction between drug exposure, multiple drug use, and the psychosocial conditions in which these infants live is impossible to unravel and control in the current investigation. Nevertheless, these confounds might be expected to maximize, rather than lessen, any differences if they existed. If anything, the differences between the two groups should have exaggerated the sleep differences, made them very large, but no such sleep differences were found.

The lack of effects in the exposed subjects might also be related to the variable amounts of substance use and the different exposure times among the mothers. It is important to recall that the mothers in this study, as is often the case, were poly-substance users, making impossible simple conclusions about any particular drug. Different substances may well have different effects. Indeed, this is confirmed by Scher et al.'s (2000) report that some substances appeared to influence infant sleep while others did not. The substance abuse history obtained through self-report also confounds the reliability of the information. Another possible explanation for the absence of sleep-wake state group differences is that in selecting a sample of infants with medically uneventful postpartum courses, those infants that were most vulnerable to the deleterious consequences of substance exposure may have been excluded. Similarly, mothers who seek treatment for their substance abuse problem, possibly to maintain custody of their infants, may differ from mothers who do not seek treatment. Finally, there may be sleep-wake effects that do not become apparent until after the first year of life. More research with improved compliance, decreased attrition, larger sample size, and better matching to the comparison group is needed.

It is clear that more longitudinal research, with larger and better-controlled samples, is required in order to better understand the mechanisms underlying sleep-wake state organization in substance-exposed infants. In addition, future research should consider giving incentives to participating families, following them at more closely-spaced ages, and/or keeping in more regular phone contact in order to help curb the high attrition rates with high-risk populations. Nevertheless, the current study, though overall seems to largely demonstrate the strong resilience of sleep organization, also somewhat supports previous studies that have reported increased fragmentation of sleep and shortened total sleep times in this population of infants.

References

Anders, T. F., Keener, M., Bowe, T. R., & Shoaff, B. A. (1982). A longitudinal study of nighttime sleep-wake patterns in infants from birth to one year. In J. O. Call, E. Galenson, & R. L. Tyson (Eds.), *Frontiers in infant psychiatry* (pp. 150-170). New York: Basic Books.

Anders, T., & Keener, M. (1985). Developmental course of nighttime sleep-wake patterns in full-term and pre-term infants during the first year of life: I. *Sleep*, *8*(3), 173-192.

Anders, T., Keener, M., & Kraemer, H. (1985). Sleep-wake state organization, neonatal assessment and development in premature infants during the first year of life: II. *Sleep, 8*(3), 193-206.

Anders, T., & Sostek, A. (1976). The use of time lapse video recording of sleep-wake behavior in human infants. *Psychophysiology, 13,* 155-158.

Anders, T., & Taylor, T. (1994). Babies and their sleep environment. *Children's Environments, 11*(2), 123-134.

Brennan, P., Grekin, E., & Mednick, S. (1999). Maternal smoking during pregnancy and adult male criminal outcomes. *Archives of General Psychiatry, 56,* 215-224.

Chasnoff, I. (1993). Missing pieces of the puzzle. *Neurotoxicity and Teratology, 15,* 287-288.

Chiriboga, C., Brust, J., Bateman, D., & Hauser, W. A. (1999). Dose-response effect of fetal cocaine exposure on newborn neurologic function. *Pediatrics, 103,* 79-85.

Church, M. (1993). Does cocaine cause birth defects? *Neurotoxicity and Teratology, 15,* 289-295.

Coles, C., Platzman, K., Smith, I., James, M. E., & Falek, A. (1992). Effects of cocaine and alcohol use in pregnancy on neonatal growth and neurobehavioral status. *Neurotoxicity and Teratology, 14,* 23-33.

Coons, S. (1987). Development of sleep and wakefulness during the first 6 months of life. In C. Guilleminault (Ed.), *Sleep disorders in children* (pp. 17-27). New York: Raven Press.

Corwin, M., Lester, B., Sepkoski, C., McLaughlin, S., Kayne, H., & Golub, H. L. (1992). Effects of in-utero cocaine exposure on newborn acoustical cry characteristics. *Pediatrics, 89,* 1199-1203.

Dahl, R. (1998). The development and disorders of sleep. *Advances in Pediatrics, 45,* 73-90.

Delaney-Black, V., Covington, C., Ostrea, E., Romero, A., Baker, D., Tagle, M. T., Nordstrom-Klee, B., Silvestre, M. A., Angelilli, M. L., Hack, C., & Long, J. (1996). Prenatal cocaine and neonatal outcome: Evaluation of dose-response relationship. *Pediatrics, 98,* 735-740.

Delaney-Black, V., Covington, C., Templin, T., Ager, J., Martier, S., & Sokol, R. (1998). Prenatal cocaine exposure and child behavior. *Pediatrics, 102,* 945-950.

Doberczak, T., Snanzer, S., Senie, R., & Kandall, S. R. (1988). Neonatal neurologic and electroencephalographic effects of intrauterine cocaine exposure. *Journal of Pediatrics, 113,* 254-258.

Dow-Edwards, D. (1993). The puzzle of cocaine's effects following maternal use during pregnancy: still unsolved. *Neurotoxicology and Teratology, 15,* 295-296.

Eisen, L., Field, T., Bandstra, E., Roberts, J. P., Morrow, C., Larson, S. K., & Steele, B. M. (1991). Perinatal cocaine effects on neonatal stress behavior and performance on the Brazelton scale. *Pediatrics, 88,* 477-480.

Eyler, F., & Behnke, M. (1999). Early development of infants exposed to drugs prenatally. *Clinics in Perinatology, 26,* 107-149.

Gingras, J., Feibel, J., Dalley, L., Meulenaer, A., & Knight, C. G. (1995). Maternal polydrug use including cocaine and postnatal infant sleep architecture: preliminary observations and implications for respiratory control and behavior. *Early Human Development, 43,* 197-204.

Joffe, S., & Chernick, V. (1990). Prediction of subsequent motor and mental retardation in newborn infants exposed to alcohol in utero by computerized EEG analysis. *Neuropediatrics, 21,* 11-17.

Johnson, J., & Leff, M. (1999). Children of substance abusers: Overview of research findings. *Pediatrics, 103,* 1085-1099.

Lester, B., Boukydis, C., & Twomey, J. (2000). Maternal substance abuse and child outcome. In C. H. Zeanah (Ed.), *Handbook of infant mental health* (2nd ed., pp. 161-175). New York: Guilford.

Lester, B., & Dreher, M. (1989). Effects of marijuana use during pregnancy on newborn cry. *Child Development, 60,* 765-771.

Lozoff, B., Askew, G. L., & Wolf, A. W. (1996). Cosleeping and early childhood sleep problems: Effects of ethnicity and socioeconomic status. *Journal of Developmental & Behavioral Pediatrics,* 17, 9-15.

Malanga, C., & Kosofsky, B. (1999). Mechanisms of action of drugs of abuse on the developing brain. *Clinics in Perinatology, 26,* 17-33.

Mayes, L., Granger, R., Frank, M., Schottenfeld, R., & Bornstein, M. H. (1993). Neurobehavioral profiles of neonates exposed to cocaine prenatally. *Pediatrics, 91,* 778-783.

Milberger, S., Biederman, J., Faraone, S., Chen, L., & Jones, J. (1996). Is maternal smoking during pregnancy a risk factor for attention deficit hyperactivity disorder in children? *American Journal of Psychiatry, 153*(9), 1138-1142.

National Institute of Drug Abuse (1996). *National pregnancy and health survey* (DHHS Publication No. 96-3819). Washington, DC: U.S. Government Printing Office.

Orlebke, J., Knol, D., & Verhulst, F. (1997). Increase in child behavior problems resulting from maternal smoking during pregnancy. *Archives of Environmental Health, 52*(4), 317-321.

Richardson, G., & Day, N. (1991). Maternal and neonatal effects of moderate cocaine use during pregnancy. *Neurotoxicity and Teratology, 13,* 455-461.

Rosett, H. L., Snyder, P., Sander, L. Lee, A., Cook, P., Weiner, L., & Gould, J. (1979). Effects of maternal drinking on neonate state regulation. *Developmental Medicine & Child Neurology, 21,* 464-473.

Scher, M., Richardson, G. A., Coble, P. A., Day, N. L., & Stoffer, D. S. (1988). The effects of prenatal alcohol and marijuana exposure: Disturbances in neonatal sleep cycling and arousal. *Pediatric Research, 24,* 101-105.

Scher, M., Richardson, G., & Day, N. (2000). Effects of prenatal cocaine/crack and other drug exposure on electroencephalographic sleep studies at birth and one year. *Pediatrics, 105,* 39-48.

Schneider, J., & Chasnoff, I. (1992). Motor assessment of cocaine/polydrug exposed infants at age 4 months. *Neurotoxicity and Teratology, 14,* 97-101.

Stewart, J., & Meeker, J. (1997). Fetal and infant deaths associated with maternal methamphetamine abuse. *Journal of Analytical Toxicology, 21,* 515-517.

Streissguth, A., Barr, H. M., Sampson, P. D., Darby, B. L., & Martin, D. C. (1989). IQ at age 4 in relation to maternal alcohol use and smoking during pregnancy. *Developmental Psychology, 25,* 3-11.

Tronick, E., & Beeghly, M. (1999). Prenatal cocaine exposure, child development, and the compromising effects of cumulative risk. *Clinics in Perinatology, 26,* 151-169.

Tronick, E. Z., Frank, D. A., Cabral, H., Mirochnick, M., & Zuckerman, B. (1996). Late dose-response effects of prenatal cocaine exposure on newborn neurobehavioral performance. *Pediatrics, 98,* 76-83.

Volpe, J. (1992). Effects of cocaine use on the fetus. *New England Journal of Medicine, 327,* 399-407.

Zuckerman, B., Frank, D., Hingson, R., Amaro, H., Levenson, S. M., Kayne, H., Parker, S., Vinci, R., Aboagye, K., Fried, L. E., et al. (1989). Effects of maternal marijuana and cocaine use on fetal growth. *New England Journal of Medicine, 320,* 762-768.

Table 1

Comparison of Longest Sleep Period and Total Sleep Time Means (SDs) between the High-Risk and Control Groups at 5 Ages

	Longest Sleep Period (min)		Total Sleep Time (min)	
	High-Risk	Control	High-Risk	Control
1 month	179 (94)	204 (61)	369 (115)	388 (99)
3 months	257 (123)	334 (103)	416 (95)	505 (65)*
6 months	194 (81)	321 (130)*	385 (108)	485 (97)*
9 months	229 (132)	289 (90)	417 (192)	491 (86)
12 months	241 (74)	326 (114)	393 (130)	522 (80)*

Note. Sample size at each age differed due to attrition. For the high risk group, at 1 month, n = 14; 3 months, n = 11; 6 months, n = 9; 9 months, n = 5; and 12 months, n = 6. The control group contained 17 infants at each age.

*$p < .05$