Published in final edited form as:
Alcohol. 2001 November ; 25(3): 153–158.

Sleep disturbances after acute exposure to alcohol in mothers' milk

Julie A. Mennella* and **Pamela L. Garcia-Gomez**
Monell Chemical Senses Center, 3500 Market Street, Philadelphia, PA 19104-3308, USA

Abstract

The results of previous research in our laboratory revealed that breast-fed infants experience significantly less active sleep after exposure to alcohol in their mothers' milk than do breast-fed infants not exposed to alcohol. The present study tested the hypothesis that infants would compensate for such reductions if their mothers then refrained from drinking alcohol. To this end, 23 breast-fed infants from 3 to 5 months of age and their mothers were tested on 2 days separated by 1 week. A small, computerized movement detector, an actigraph, was placed on the infants' left ankles to monitor sleep and activity patterning after which they were bottle fed mother's milk alone (control condition) on 1 test day and mother's milk containing 32 mg of ethanol per 100 ml — the average concentration detected in human milk after lactating women drank an acute dose (0.3 g/kg) of alcohol — on the other. The infants' behaviors were monitored for the next 24 h; the first 3.5 h of monitoring on each test day took place at the Monell Center. Consistent with previous findings, infants exhibited significantly less active sleep during the 3.5 h immediately after exposure to alcohol in mothers' milk compared with the control condition; the decrease in active sleep was observed in all but 4 of the infants tested. Compensatory increases in active sleep were then observed in the next 20.5 h, when mothers refrained from drinking alcohol. Although the mechanisms underlying the reduction in sleep remain to be elucidated, these findings demonstrate that short-term exposure to small amounts of alcohol in mothers' milk produces distinctive changes in the infants' sleep–wake patterning.

Keywords

Alcohol; Lactation; Sleep; Activity; Infant behavior

1. Introduction

Because of the striking paucity of scientific investigations of alcohol and breast feeding, women, and consequently their infants, have had to rely on a rich folklore that has been passed down from generations (see Fildes, 1986). This lore relates that alcohol has galactogenic properties that facilitate milk let down and rectify milk insufficiency and has sedative properties that alleviate and calm the "fussy" infant (Adams & Davidson, 1987; Auerbach et al., 1987; Grossman, 1988; Lawrence, 1989; Mennella, 1999). Such advice is remarkably reminiscent of that given to pregnant women about alcohol use before the scientific and clinical investigations of prenatal alcohol exposure (Davidson et al., 1981). That is, there are no dangers and it is good for you and your baby! Such beliefs continue to be ingrained in current medical practice. Although some women are advised to abstain from drinking during pregnancy, Mulder and colleagues (1998) report that, in Britain, women are often told that drinking one to two drinks once or twice a week is unlikely to affect fetal development (Dillner et al., 1996). Similarly,

*Corresponding author. Tel.: +1-215-898-9230; fax: +1-215-898-2084. mennella@monell.org (J.A. Mennella).
Editor: S. Borg

the results of a recent study revealed that 25% of the 410 lactating women surveyed in the Delaware Valley reported that they were encouraged by a health professional to drink alcohol to improve the quality, quantity, and flow of their milk or to help their babies get a good night's sleep (Mennella, 2002).

Research has revealed that acute exposure to alcohol in mothers' milk altered the infants' sleep–wake patterning in ways that are contrary to this medical lore (Mennella & Beauchamp, 1991; Mennella & Gerrish, 1998). Infants, whose mothers drank little during both pregnancy and lactation, slept for significantly shorter periods during the hours immediately after the consumption of alcohol in their mothers' milk compared with their being fed mothers' milk containing no alcohol (Mennella & Gerrish, 1998). This reduction was due, in part, to a shortening in the amount of time that the infants spent in active sleep, a finding consistent with that observed in the near-term fetus (Mulder et al., 1998), in normal adults (Rundell et al., 1972; Williams et al., 1983), and in other animals (Mendelson & Hill, 1978) after alcohol exposure.

The goals of the present study were to replicate our previous findings that infants exposed to alcohol in their mothers' milk have altered sleep patterns and to test the hypothesis that breast-fed infants will compensate for the disruptions in active sleep that occur after exposure to alcohol in their mothers' milk if their mothers then refrain from drinking alcohol. That they are capable of such compensation is suggested by the observation that newborns whose mothers drank heavily throughout pregnancy spent a greater proportion of time in both quiet and active sleep during the immediate postpartum period if they were predominantly formula fed after birth (Chernick et al., 1983; Rosett et al., 1979; Scher et al., 1988). Of additional interest is the finding that day-old infants who were manually awakened during sleep exhibited a recovery of sleep after such sleep deprivation (Anders & Roffwarg, 1973).

2. Methods

2.1. Subjects

Twenty-three nonsmoking, lactating women who had consumed at least one alcoholic beverage during lactation and who had infants with experience in drinking human milk from a bottle were recruited from ads in local newspapers and from the Women, Infants, and Children (WIC) centers in Philadelphia, PA. The mothers (82.6% multiparous) were 32.7 ± 1.2 years of age, and their infants (13 girls, 10 boys) ranged in age from 3.1 to 5.1 months (mean = 4.0 ± 0.1 months). None of the infants or mothers were taking any medication that would have affected sleep. Four additional mother–infant pairs began testing but were excluded because the infants would not accept the bottle. Informed consent was obtained from each woman before testing began. All procedures used in this study were approved by the Committee on Studies Involving Human Beings at the University of Pennsylvania.

At the end of testing, mothers were interviewed and asked to complete a series of questionnaires that elicited such information as the type of advice, if any, given to them about alcohol use during lactation. They also estimated the number, types, and frequency of alcoholic beverages consumed during pregnancy and lactation by completing a time-line, follow-back questionnaire (Sokol et al., 1981). All but one of the mothers reported drinking very little during pregnancy (range = 0–16 alcoholic beverages per month; mean = 1.5 ± 0.7 drinks per month) and slightly, but not significantly, increasing alcohol intake during lactation to, on average, 4.9 ± 2.1 alcoholic beverages per month [range = < 1–50 drinks per month; paired $t(22\text{df}) = -1.75$; $P = .09$]. Approximately one-third (34.8%) of the mothers reported drinking at least one alcoholic beverage in the 2 weeks preceding their participation in the study. In addition, approximately 35% of the mothers reported that they were advised to drink alcohol during lactation by a health professional to facilitate lactation, to help their babies sleep better, or both,

whereas the remaining women were not given any advice about drinking. Similar findings were obtained when we assessed the type of advice given by family members and friends (34.8% were encouraged to drink; 17.4% were discouraged, whereas the remaining received no advice).

2.2. Procedures

A within-subjects design was implemented. Each mother–infant pair was tested at the Monell Center on 2 days separated by an interval of approximately 1 week (see Mennella & Gerrish, 1998, for more details). Mothers were instructed to refrain from drinking any alcoholic beverages on the 3 days before and the 2 days after each test day. Each mother arrived with her infant at the Monell Center at approximately 9:30 a.m., having last fed her infant at approximately the same time on each testing day [paired $t(22df) = -1.06$; $P = .30$]. Testing took place in a private, carpeted room containing a portable crib for the infants. After acclimatization to the room and personnel, each mother expressed approximately 100 ml of milk, usually from both breasts, by using an electric breast pump (Medela, Crystal Lake, IL), and an actigraph was placed on each infant's left leg.

After the actigraph had been in place for approximately 15 min, the infants were bottle fed approximately 100 ml of their mothers' milk alone on one test day and an equal volume of their mothers' milk containing 32 mg of ethanol on the other test day; this amount of ethanol is the average concentration detected in human milk approximately 1 h after lactating women drank an acute dose (0.3 g/kg) of alcohol (Mennella & Beauchamp, 1991, 1993). The infants were fed their mothers' milk from a bottle because previous research revealed that intake is diminished when infants feed at the breast after maternal alcohol consumption (Mennella & Beauchamp, 1991, 1993). Eleven of the infants were fed the control milk on the first and the alcohol-containing milk on the second test day; the order was reversed for the remaining infants. There were no significant effects of order of testing or of sex of the infant on any of the variables tested.

The infants were monitored for at least 24 h after they consumed the breast milk from the bottle. The first 3.5 h of monitoring on both testing days took place at the Monell Center, after which each mother–infant pair immediately returned home with the actigraph attached to the infant's leg. Throughout the day, the mothers nursed their infants on demand; milk intake, during the first 3.5 h of testing on both days, was assessed by weighing the infants immediately before and after each feed on an Acme Medical Scale (San Leandro, CA), which was accurate to 5.0 g. The infants were always placed prone in a crib or on the floor while sleeping because the actigraph-monitored infant activity must be independent of their mothers' activity and because body position influences sleep patterning in infants (Kahn et al., 1993; Sahni et al., 1999). Mothers were contacted by telephone to ensure compliance. The mothers were not informed of the order of testing, and at the end of each test session they were asked several questions about their infants' behavior.

2.3. Dose delivered to the infant

The amount of alcohol ingested by the infants in this study (estimated by multiplying the volume of milk ingested by the concentration of alcohol) ranged from 24.3 to 32.0 mg (mean = 30.5 ± 0.3 mg). With the body weight of each infant taken into account, the estimated dose ranged from 3.5 to 5.9 mg/kg (mean = 4.5 ± 0.1 mg/kg); this amount is similar to that ingested at the breast after the consumption of a 0.3-g/kg dose by the mother (Mennella & Beauchamp, 1991, 1993).

2.4. Method for measuring infant sleep and activity rhythms

The actigraph (AMA-32 Ambulatory Monitoring, Ardsley, NY), a self-contained microcomputer consisting of a piezoelectric accelerometer, generates a voltage in proportion to the mechanical deflection of the free end as the actigraph is moved (Sadeh et al., 1989, 1991, 1995). Motility levels were sampled in the zero-crossing mode at a constant rate of 10 Hz. In this mode, an activity count was scored each time that an infant's leg movement fell above the unit's sensitivity threshold. The number of zero crossings was stored in the actigraph's memory in 1-min epochs and later analyzed with the use of a computer program (Ambulatory Monitoring Ltd., Ardsley, NY). The automatic scoring algorithm on which this program is based has been validated by a behavioral observational state taxonomy assessment of sleep stages in infants of this age and can reliably distinguish between active and quiet sleep (Sadeh et al., 1995). From the raw activity data, the program determined the number of minutes spent in active and quiet sleep, the number of sleeping bouts, and the mean activity count (average number of zero crossings of the piezoelectric beam) during wakefulness that occurred during each 3.5-h (0–3.5 h) test session for each infant and then for the 20.5 h (3.5–24 h) that followed each 3.5-h test session for all but one of the infants, whose assessment was compromised by technical difficulties.

2.5. Data analyses

Repeated measures analyses of variance (2 × 2 repeated measures ANOVAs) were conducted to determine whether there were significant differences in these measures as a function of time since exposure (0–3.5, 3.5–24 h postexposure) and day of the experimental period (control, alcohol). To allow for comparisons between the two time periods (e.g., 0–3.5 h versus 3.5–24 h) for the interval data, we determined the hourly rate for each measure (e.g., minutes per hour, number of bouts per hour), with the exception of mean activity count during wakefulness, which is expressed as the average number of zero crossings of the piezoelectric beam in each time period. Significant effects in the ANOVA were probed by paired *t* tests. All summary statistics reported in this article are expressed as means ± S.E.M., and all *P* values represent two-tailed tests.

3. Results

There was a significant interaction between the time since exposure (0–3.5 vs. 3.5–24 h) and the experimental test day (i.e., control, alcohol) for the amount of time that infants spent in active sleep [$F(1,21df) = 14.1$; $P = .001$]. Consistent with previous findings (Mennella & Gerrish, 1998), infants spent less time in active sleep [paired $t(22df) = 2.11$; $P = .05$] during the hours immediately after exposure to alcohol in their mothers' milk (Table 1). A decrease in active sleep was observed in 19 of the 23 infants.

Also consistent with previous findings (Mennella & Gerrish, 1998), the effects of alcohol exposure on active sleep were not immediate [$F(1,22df) = 8.68$; $P = .007$]. As seen in Fig. 1, there was no significant difference in the amount of time spent in active sleep during the first half of the 3.5-h testing sessions [paired $t(22df) = -0.88$; $P = .39$; not significant]. However, infants spent significantly less time in active sleep during the second half of the test session (i.e., 1.75–3.5 h), in which they were fed alcohol in mothers' milk, compared with being given mothers' milk containing no alcohol [paired $t(22df) = 3.68$; $P = .001$]. The data also revealed that infants compensated for such decreases when their mothers then refrained from drinking alcohol [paired $t(21df) = -2.73$; $P = .01$]. On average, infants exhibited a 22.4 ± 7.0% increase in active sleep during the 20.5 h after the test session in which they were exposed to alcohol compared with when they were exposed to mothers' milk containing no alcohol (Fig. 2). There were no significant interactions between time since exposure (0–3.5 vs. 3.5–24 h) and experimental test day (i.e., control, alcohol) for any of the other variables tested (e.g., longest

sleep bout, total sleep, quiet sleep, number of bouts, activity during wakefulness; all values of $P > .10$).

Mothers were apparently unaware of the differences in their infants' behaviors after alcohol exposure. That is, they were as likely as not to report that they thought that their infants consumed the alcohol-containing milk on either test day (Fisher exact probability test; $P = 1.0$; not significant). Moreover, there were no significant differences in the number of times that the infants breast fed [control vs. alcohol: 2.4 ± 0.3 vs. 2.5 ± 0.2; paired $t(22df) = 0.42$; $P = .68$; not significant] or in the average amount of breast milk consumed during each feed [control vs. alcohol: 69.9 ± 8.0 vs. 70.2 ± 5.5 ml; paired $t(22df) = 0.03$; $P = .97$; not significant]. As a preliminary step in determining whether maternal beliefs affected the infants' responses to alcohol exposure, an ANOVA was conducted to determine whether there were differences between infants whose mothers were encouraged to drink alcohol during lactation by a health professional (35%) and those whose mothers received no advice at all (65%). There were no significant effects between these two groups on the infants' responses to alcohol for any of the sleep or activity measures studied [e.g., active sleep: $F(1,20df) = 0.27$; $P = .61$)].

4. Discussion

The findings in the present study further demonstrate that exposure to alcohol in mothers' milk alters the patterning of infant sleep. That is, breast-fed infants spent significantly less time in active sleep and tended to be less active during wakefulness in the hours immediately after exposure to alcohol in their mothers' milk. The effects of alcohol were not immediate, however, and were evidenced during the last half of the 3.5-h testing session. Such findings resemble observations made in the near-term fetus. Mulder and colleagues (1998) found that the acute consumption of two glasses of wine (dose = 0.25 g/kg) by pregnant women who drank very little or not at all during pregnancy had an effect on the fetal behavioral-state organization. Rapid eye movement sleep, in particular, was reduced, and breathing activity was suppressed.

As shown in Fig. 2, the infants then compensated for this reduction in active sleep during the 20.5 h after alcohol exposure. Recall that their mothers refrained from drinking alcohol during this period. Whether similar findings would be observed if mothers continued to drink seems unlikely. Of particular interest here is the observation that the fetus can compensate, in part, for sleep disturbances induced by alcohol exposure in utero if they are not additionally exposed to alcohol after birth. That is, infants of mothers who drank heavily throughout pregnancy spent a greater proportion of time in both quiet and active sleep during the immediate postpartum period if they were predominantly formula fed after birth, compared with infants of mothers who abstained from drinking (Chernick et al., 1983;Rosett et al., 1979;Scher et al., 1988). Compensatory increases in active sleep in the later part of the night after the acute consumption of alcohol by nonalcoholic mothers also have been reported (Rundell et al., 1972;Williams et al., 1983;Yules et al., 1966), and although tolerance to the sleep-disruptive effects of alcohol develops within a few nights, compensatory increases in active sleep recur when alcohol is discontinued.

Sleep, the most frequent state of consciousness of infants, can be influenced by a variety of environmental and physiological factors. The present study aimed to experimentally control a variety of such factors. For example, each of the test sessions was held at the same time of day, and testing took place in a private, quiet room. The infants were placed prone in a crib or on a carpet as much as possible, so that the infants' actigraph-monitored sleep and activity measures were independent of their mothers' activity (Kahn et al., 1993; Sahni et al., 1999). The mothers were unaware of the order of testing and did not reliably identify which day their infants consumed the alcohol-containing milk. Moreover, there was no difference in the patterning of feeding immediately before or during the two testing days, which were separated by an interval

of 1 week. And, finally, there were no significant effects of maternal beliefs, as assessed by the type of advice received by the mothers about alcohol use during lactation, on any of the sleep measures studied. Although possible, it seems unlikely that the alterations in the patterning of sleep were due to changes in the infants' interaction with their mothers. Rather, it appears that exposure to this small amount of alcohol in the mothers' milk had direct, albeit subtle, effects on the infants.

The mechanisms underlying these changes in sleep patterning and the long-term effect of chronic exposure on development remain to be elucidated (refer to Little et al., 1989). Because the drug in the nursing infants' blood or urine was not measured, we do not know the amount that was absorbed from the milk. However, the available information on pediatric pharmacokinetics has demonstrated significant differences in the absorption, distribution, metabolism, and excretion of a variety of drugs from those of older children and adults (Milsap & Jusko, 1994). Some evidence supports the suggestion that infants have limited capacity to oxidize ethanol (Pikkarainen & Raiha, 1967), which, in turn, may render the dose more potent. The results of animal-model studies have revealed that infant rats exhibit a lower alcohol-related metabolic capacity, longer half-life of alcohol, and, in turn, higher peak blood alcohol levels (Kelly et al., 1987), and they are more sensitive to the effects of alcohol on certain cognitive processes (Chen et al., 1992) compared with older-aged conspecifics.

The goal of our research program is to experimentally investigate the relation between alcohol and breast feeding, so that we can provide mothers and health professionals with some answers to frequently asked questions on this topic (Auerbach et al., 1987; Grossman, 1988). Unlike a pregnant woman, a nursing woman who drinks occasionally can limit her infant's exposure to alcohol by timing breast feeding in relation to her drinking. That is, alcohol is not stored in breast milk; rather, its presence parallels that found in maternal plasma, peaking approximately one-half hour to an hour after the cessation of drinking and decreasing thereafter (Lawton, 1985; Mennella & Beauchamp, 1991). Contrary to popular belief but consistent with the results of several animal-model studies (Heil & Subramanian, 2000; Oyama et al., 2000; Subramanian, 1995, [1999]; Subramanian & Abel, 1988; Swiatek et al., 1986; Vilaró et al., 1987), infants actually ingest less milk at the breast in the hours immediately after maternal alcohol consumption (Mennella & Beauchamp, 1991, 1993). This diminished intake is due, in part, to a direct effect of alcohol on milk production by the mother (Mennella, 1998). If a nursing mother is told to drink to help her baby sleep better, she and her caretakers can refer to the research, albeit limited, that calls this lore into serious question. Results of the present study, which expand on previous research findings (Mennella & Gerrish, 1998), demonstrate that infant sleep is disrupted during the 24 h after exposure to alcohol in mothers' milk.

Acknowledgments

We acknowledge the valuable comments and assistance of Dr. Carolyn Gerrish. This research was supported by grant no. AA09523 from the National Institute on Alcohol Abuse and Alcoholism and the Office of Research on Women's Health.

References

Adams LM, Davidson M. Present concepts of infant colic. Pediatr Ann 1987;16:817–820. [PubMed: 3317233]

Anders TF, Roffwarg HP. The effects of selective interruption and deprivation of sleep in the human newborn. Dev Psychobiol 1973;6:77–89. [PubMed: 4353450]

Auerbach KG, Schreiber JR, Blume S, Falkner F. Beer and the breast-feeding mom [Questions and Answers]. JAMA 1987;258:2126.

Chen WJ, Spear LP, Spear NE. Enhancement of sensory preconditioning by a moderate dose of ethanol in infant and juvenile rats. Behav Neural Biol 1992;57:44–57. [PubMed: 1567333]

Chernick V, Childiaeva R, Ioffe S. Effects of maternal alcohol intake and smoking on neonatal electroencephalogram and anthropometric measurements. Am J Obstet Gynecol 1983;146:41–47. [PubMed: 6846423]

Davidson S, Alden L, Davidson P. Changes in alcohol consumption after childbirth. J Adv Nurs 1981;6:195–198. [PubMed: 6909224]

Dillner L, Josefson D, Karcher H, Sheldon T, Dorozynski A, Zinn C. Alcohol: pushing the limits. BMJ 1996;312:7–9. [PubMed: 8555891]

Fildes, V. Breasts, Bottles and Babies: A History of Infant Feeding. Edinburgh: Edinburgh University Press; 1986.

Grossman ER. Beer, breast-feeding, and the wisdom of old wives. JAMA 1988;259:1016. [PubMed: 3339797]

Heil SH, Subramanian MG. Chronic alcohol exposure and lactation: extended observations. Alcohol 2000;21:127–132. [PubMed: 10963935]

Kahn A, Groswasser J, Sottiaux M, Rebuffat E, Franco P, Dramaix M. Prone or supine body position and sleep characteristics in infants. Pediatrics 1993;91:1112–1115. [PubMed: 8502511]

Kelly SJ, Bonthius DJ, West JR. Developmental changes in alcohol pharmacokinetics in rats. Alcohol Clin Exp Res 1987;11:281–286. [PubMed: 3307494]

Lawrence, RA. Breastfeeding: A Guide for the Medical Profession. St. Louis, MO: Mosby; 1989.

Lawton ME. Alcohol in breast milk. Aust N Z J Obstet Gynaecol 1985;25:71–73. [PubMed: 3862407]

Little RE, Anderson KW, Ervin CH, Worthington-Roberts B, Clarren SK. Maternal alcohol use during breast feeding and infant mental and motor development at one year. N Engl J Med 1989;321:425–430. [PubMed: 2761576]

Mendelson WB, Hill SY. Effects of the acute administration of ethanol on the sleep of the rat: a dose-response study. Pharmacol Biochem Behav 1978;8:723–726. [PubMed: 211533]

Mennella JA. Short-term effects of maternal alcohol consumption on lactational performance. Alcohol Clin Exp Res 1998;22:1389–1392. [PubMed: 9802517]

Mennella, JA. The transfer of alcohol to human milk: sensory implications and effects on mother-infant interaction. In: Hannigan, JH.; Spear, N.; Spear, L.; Goodlett, CR., editors. Alcohol and Alcoholism: Brain and Development. Mahwah, NJ: Lawrence Erlbaum; 1999. p. 177-198.

Mennella, JA. Alcohol use during lactation: the folklore versus the science. In: Auerbach, KG., editor. Current Issues in Clinical Lactation 2002. Sudbury, MA: Jones and Burtlett Publishers; 2002.

Mennella JA, Beauchamp GK. The transfer of alcohol to human milk: effects on flavor and the infant's behavior. N Engl J Med 1991;325:981–985. [PubMed: 1886634]

Mennella JA, Beauchamp GK. Beer, breast feeding and folklore. Dev Pychobiol 1993;26:459–466.

Mennella JA, Gerrish CJ. Effects of exposure to alcohol in mother's milk on infant sleep. Pediatrics 1998;101(5):E21–E25.

Milsap RL, Jusko WJ. Pharmacokinetics in the infant. Environ Health Perspect 1994;102:107–110. [PubMed: 7737034]

Mulder EJH, Morssink LP, van der Schee T, Visser GHA. Acute maternal alcohol consumption disrupts behavioral state organization in the near-term fetus. Pediatr Res 1998;44:774–779. [PubMed: 9803461]

Oyama LM, Couto RC, Couto GEC, Dâmaso AR, Oller do Nascimento CM. Ethanol intake during lactation. I. Effects on dams' metabolism and pups' body weight gain. Alcohol 2000;21:195–200. [PubMed: 11091021]

Pikkarainen PH, Raiha NCR. Development of alcohol dehydrogenase activity in the human liver. Pediatr Res 1967;1:165–168. [PubMed: 6080860]

Rosett HL, Snyder P, Sander LW, Lee A, Cook P, Weiner L, Gould J. Effects of maternal drinking on neonate state regulation. Dev Med Child Neurol 1979;21:464–473. [PubMed: 520694]

Rundell OH, Lester BK, Griffiths WJ, Williams HL. Alcohol and sleep in young adults. Psychopharmacologia 1972;26:201–218. [PubMed: 4342143]

Sadeh A, Acebo C, Seifer R, Aytur S, Carskadon MA. Activity-based assessment of sleep– wake patterns during the 1st year of life. Infant Behav Dev 1995;18:329–337.

Sadeh A, Alster J, Urbach D, Lavie P. Actigraphically based automatic bedtime sleep– wake scoring: validity and clinical applications. J Amb Monit 1989;2:209–216.

Sadeh A, Lavie P, Scher A, Tirosh E, Epstein R. Actigraphic home-monitoring sleep-disturbed and control infants and young children: a new method for pediatric assessment of sleep –wake patterns. Pediatrics 1991;87:494–499. [PubMed: 2011426]

Sahni R, Schulze KF, Kashyap S, Ohira-Kist K, Myers MM, Fifer WP. Body position, sleep states, and cardiorespiratory activity in developing low birth weight infants. Early Hum Dev 1999;54:197–206. [PubMed: 10321786]

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. Pediatr Res 1988;24:101–105. [PubMed: 3412843]

Sokol RJ, Miller SI, Debanne S, Golden N, Collins G, Kaplan J, Martier SS. The Cleveland NIAAA prospective alcohol-in-pregnancy study: the first year. Neurobehav Toxicol Teratol 1981;3:203–209. [PubMed: 7254465]

Subramanian MG. Effects of chronic alcohol administration on lactational performance in the rat. Alcohol 1995;12:137–143. [PubMed: 7772265]

Subramanian MG. Alcohol inhibits suckling-induced oxytocin release in the lactating rat. Alcohol 1999;19:51–55. [PubMed: 10487388]

Subramanian MG, Abel EL. Alcohol inhibits suckling-induced prolactin release and milk yield. Alcohol 1988;5:95–98. [PubMed: 3395466]

Swiatek KR, Dombrowski GJ Jr, Chao KL. The inefficient transfer of maternally fed alcohol to nursing rats. Alcohol 1986;3:169–174. [PubMed: 3741612]

Vilaró S, Viñas O, Remesar X, Herrera E. Effects of chronic ethanol consumption on lactational performance in rat: mammary gland and milk composition and pups' growth and metabolism. Pharmacol Biochem Behav 1987;27:333–339. [PubMed: 3628448]

Williams DL, MacLean AW, Cairns J. Dose-response effects of ethanol on the sleep of young women. J Stud Alcohol 1983;44:515–523. [PubMed: 6645531]

Yules RB, Feedman DX, Chandler KA. The effect of ethyl alcohol on man's electroencephalographic sleep cycle. Electroencephalogr Clin Neurophysiol 1966;20:109–111. [PubMed: 4159908]

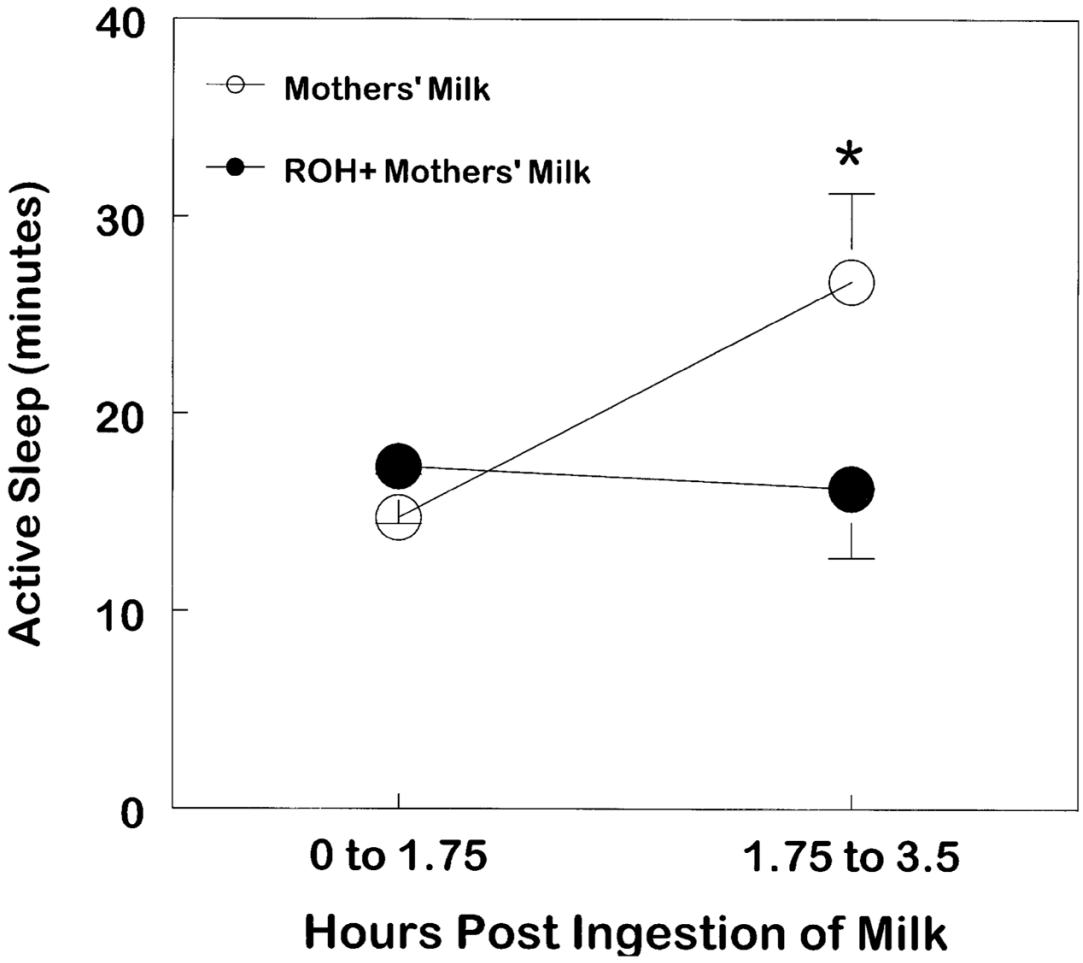

Fig. 1.

Mean number of minutes that the infants spent in active sleep during the first and second half of the 3.5-h testing session in which they consumed mothers' milk not containing alcohol (open circles) or mothers' milk containing ethanol (closed circles) [$F(1,22df) = 8.68$; $P = .007$]. ROH = alcohol.

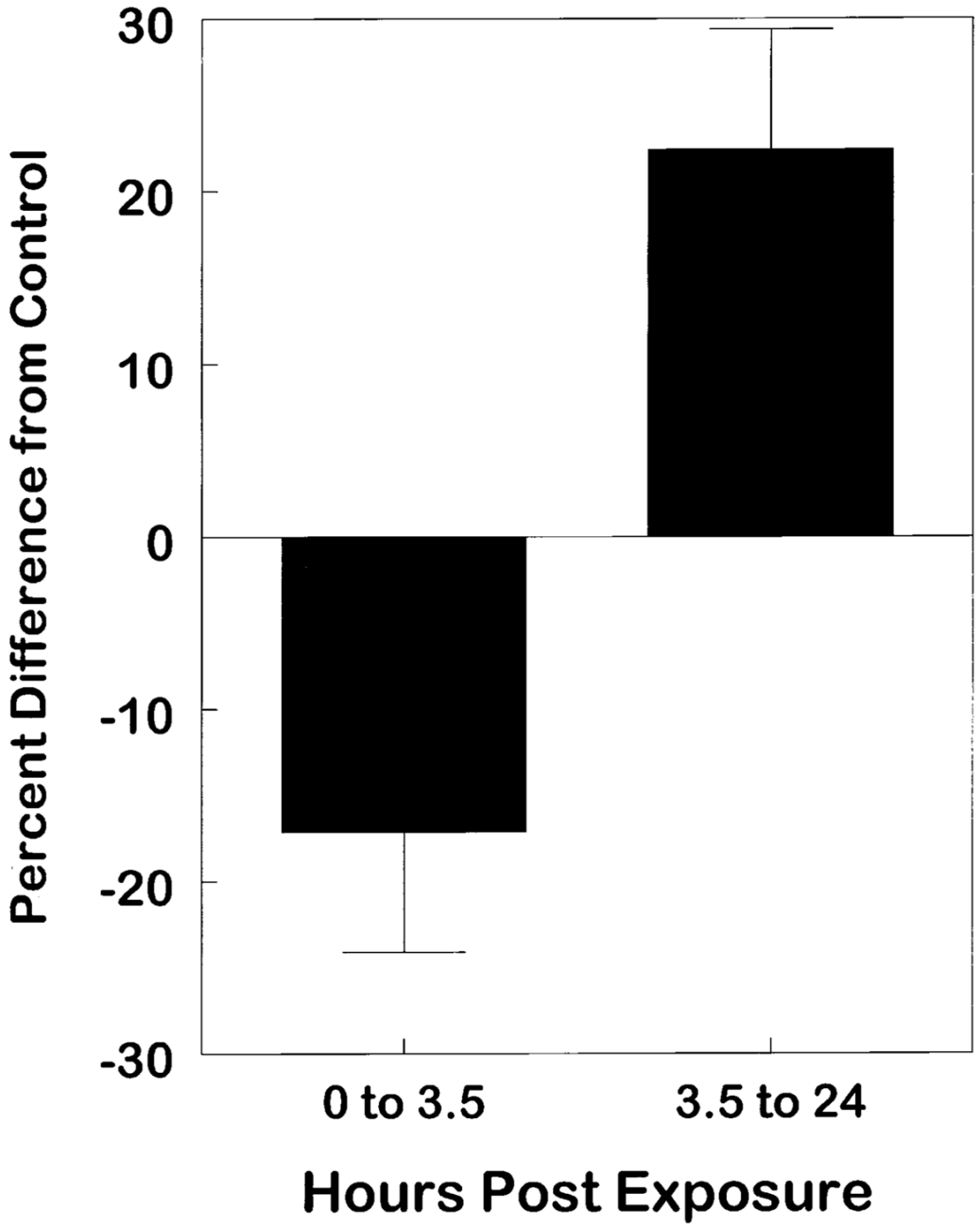

Fig. 2.

Mean percentage difference in the time that infants spent in active sleep during the first 3.5 h (0–3.5 h) and then the following 20.5 h (3.5–24 h) in which the infants were bottle fed approximately 100 ml of their mothers' milk containing ethanol compared with when they were bottle fed mothers' milk not containing alcohol.

Table 1

Short-term effects of exposure to alcohol in mother's milk on sleep and activity levels

	Time since feeding			
	0 to 3.5 h		3.5 to 24 h	
	Milk only	Alcohol + milk	Milk only	Alcohol + milk
Total sleep (min)	72.2 ± 7.4	66.6 ± 9.1	675.7 ± 25.3	711.8 ± 21.9
Quiet sleep (min)	30.7 ± 4.0	33.0 ± 5.2	346.3 ± 24.7	326.4 ± 16.1
Active sleep (min)	41.5 ± 5.4	33.5 ± 5.3*	329.4 ± 20.0	385.4 ± 22.6*
Longest sleep bout (min)	55.5 ± 6.1	51.7 ± 7.5	301.9 ± 27.4	340.5 ± 29.7
Number of sleeping bouts	2.3 ± 0.2	2.2 ± 0.3	9.9 ± 0.6	10.5 ± 0.8
Mean activity count during wakefulness	218.1 ± 4.1	209.0 ± 6.1	210.7 ± 3.5	212.5 ± 4.7

Data are presented for the first 3.5 h after exposure and then the next 20.5 h. On one testing day, the infants were bottle fed mothers' milk only, whereas, on the other, they were fed mothers' milk containing alcohol.

* $P \leq .05$ compared with milk only.