

Sleep disturbances after acute exposure to alcohol in mothers' milk

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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. © 2002 Elsevier Science Inc. All rights reserved.

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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, 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

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(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 $t(22df) = -1.75$; $P = .09$]. 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

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.

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 $22.4 \pm 7.0\%$ 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

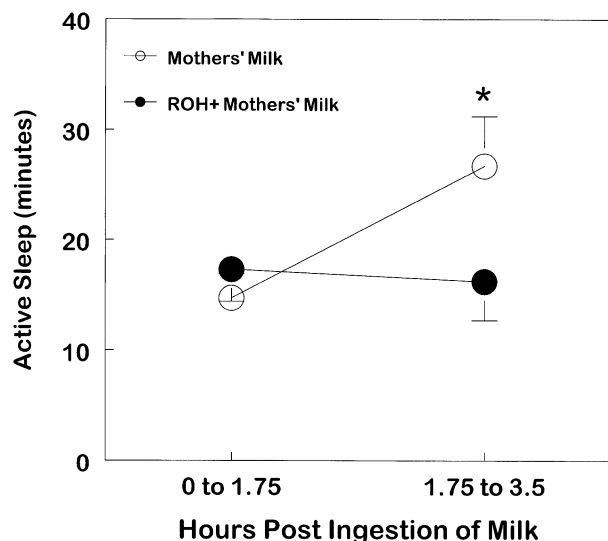


Fig. 1. Mean number of minutes that the infants spent in active sleep during the first and second half of the 3.5-h testing session in which they consumed mothers' milk not containing alcohol (open circles) or mothers' milk containing ethanol (closed circles) [$F(1,22df) = 8.68$; $P = .007$]. ROH = alcohol.

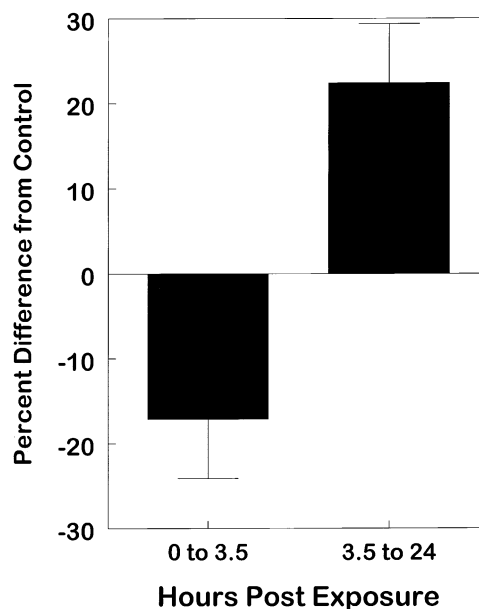


Fig. 2. Mean percentage difference in the time that infants spent in active sleep during the first 3.5 h (0–3.5 h) and then the following 20.5 h (3.5–24 h) in which the infants were bottle fed approximately 100 ml of their mothers' milk containing ethanol compared with when they were bottle fed mothers' milk not containing alcohol.

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.

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