

Effects of Exposure to Alcohol in Mother's Milk on Infant Sleep

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ABSTRACT. *Objective.* To test the hypothesis that exposure to alcohol in breast milk affects infants' sleep and activity levels in the short term.

Methods. Thirteen lactating women and their infants were tested on 2 days, separated by an interval of 1 week. On each testing day, the mother expressed 100 mL of milk, while a small, computerized movement detector called an actigraph was placed on the infant's left leg to monitor sleep and activity patterning. After the actigraph had been in place for ~15 minutes, the infants ingested their mother's breast milk flavored with alcohol (32 mg) on one testing day and breast milk alone on the other. The infants' behaviors were monitored for the next 3.5 hours.

Results. The infants spent significantly less time sleeping during the 3.5 hours after consuming the alcohol-flavored milk (78.2 minutes compared with 56.8 minutes after feeding alcohol in breast milk). This reduction was apparently attributable to a shortening in the longest sleeping bout (34.5 compared with 56.7 minutes for sleeping after breast milk alone) and the amount of time spent in active sleep (25.8 minutes compared with 44.2 minutes after breast milk alone); the decrease in active sleep was observed in all but 2 of the 13 infants tested.

Conclusions. Although the mechanisms underlying the reduction in sleep remain to be elucidated, this study shows that short-term exposure to small amounts of alcohol in breast milk produces distinctive changes in the infant's sleep-wake patterning. *Pediatrics* 1998;101(5). URL: <http://www.pediatrics.org/cgi/content/full/101/5/e2>; alcohol, lactation, sleep, activity, development, infant behavior.

ABBREVIATION. NS, not significant.

The notion that infants can be influenced by components in their mothers' breast milk has a rich, long history.¹ Physicians in the 18th century often would treat the infants' ailments by prescribing certain diets to the mother or wet nurse.² That such practices continue in more modern times is evident in the medical lore that recommends that nursing mothers drink an occasional alcoholic beverage, preferably before the evening feeding, to alleviate and sedate their fussy infants.^{3,4} No experimental evidence supports this recommendation, although a previous study on the effects of maternal alcohol consumption on the feeding behaviors of breastfed infants reported that the infants' patterning

of sleeping, as determined from logs completed by mothers, indeed was altered in the short term.⁵

Despite this lore, the question of whether occasional exposure to alcohol in breast milk can affect the infant continues to generate much speculation in the medical community.^{6,7} Because alcohol is excreted to a limited extent in breast milk,^{5,8,9} occasional exposure often is considered insignificant,^{8,10} except in such rare cases of intoxication when the mothers of breastfeeding infants drank quite heavily^{11,12} or when infants were inadvertently fed large amounts of alcohol in a bottle.¹³ Moreover, an epidemiologic study¹⁴ found no significant difference in the motor and mental development of 1-year-old breastfed infants whose mothers drank less than one drink per day, compared with either breastfed infants whose mothers did not drink at all or those who were formula-fed. Only those infants who were exposed regularly (at least daily) to alcohol in their mothers' breast milk, showed a slight, but significant deficit in gross motor, but not mental, development. Perhaps the developing brain is extremely sensitive to alcohol or the small amounts ingested in breast milk accumulated in the infant because it is metabolized or excreted more slowly than in children and adults.¹⁴

The present study aimed to evaluate the infants' sleep and activity levels during the immediate hours after acute exposure to alcohol in breast milk. Because of the limitations in mothers' reports,¹⁵ objective measures were obtained by using a miniature actigraph, a small, lightweight wristwatch-sized motion detector, that had been validated previously with polysomnographic monitoring of both infants¹⁶ and adults.¹⁷

METHODS

Subjects

Fifteen nonsmoking lactating women who had consumed at least one alcoholic beverage during lactation and whose infants had experienced drinking breast milk from a bottle were recruited from advertisements in local newspapers and from the Women, Infant and Children Centers in Philadelphia, PA. Two of the mother-infant pairs were excluded because one infant had a fever on a testing day and the other received a vaccination injection the day before testing and cried throughout the session. The mothers (3 primiparous, 10 multiparous) ranged in age from 22 to 34 years (mean, 27.4 ± 1.1 years), and the infants (9 girls, 4 boys) ranged in age from 1.5 to 5.6 months of age (mean, 2.7 ± 0.3 months). There was no effect of the infants' sex on any of the variables tested. Informed consent was obtained from each woman before testing. All procedures used in this study were approved by the Committee on Studies Involving Human Beings at the University of Pennsylvania.

Using a time-line follow-back questionnaire,¹⁸ each woman estimated the number, types, and frequency of alcoholic beverages consumed during pregnancy and lactation. Mothers reported

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drinking very little during pregnancy (range, 0 to 30 alcoholic beverages per 9 months; mean \pm SEM, 1.3 ± 1.1), but increasing alcohol intake during lactation, on average, to 3.0 ± 1.0 alcoholic beverages per month (range, <1 to 20 drinks per month); these numbers likely underestimate alcohol usage.¹⁹ At the end of the study, mothers were asked to refrain from drinking one alcoholic beverage in the near future so that their infant would not be additionally exposed to alcohol as a result of their participation in the study.

Procedures

Each mother–infant pair was tested on 2 days separated by an interval of 1 week (\pm 1 day). During the 3 days preceding each testing day, mothers were instructed to eat bland foods and to refrain from drinking any alcoholic beverages to ensure that the milk would have a similar flavor profile and be devoid of alcohol on each testing day.²⁰ Testing occurred in a private room that was carpeted and contained 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 electronic breast pump (Medela, Crystal Lake, IL), and the actigraph was placed on the infant's left leg.

After the actigraph had been in place for \sim 15 minutes, each infant readily bottle-fed \sim 100 mL of their mothers' breast milk alone on one testing day and an equal volume of their mothers' breast milk flavored with 40 μ L (32 mg) of ethanol on the other test day; the amount of ethanol added to the milk represents the average concentration detected in breast milk \sim 1 hour after lactating women drank an acute dose (0.3 g/kg) of alcohol.^{5,8,9,21} Seven of the infants were fed the control milk on the first day and the alcohol-flavored milk on second test day; the order was reversed for the remaining infants. There were no significant effects of order of presentation on any of the variables tested.

The infants were monitored for 3.5 hours after they consumed the breast milk from the bottle. The mothers nursed their infants on demand; milk intake was assessed by weighing the infants immediately before and after each feed on an Acme medical scale (San Leandro, CA) that was accurate to 5.0 g. The volume of milk consumed by the infant (in milliliters) was estimated by dividing the weight of the milk by 1.031, the specific gravity of mature breast milk. The infants were placed in the portable crib provided or on the carpet as much as possible so that actigraph-monitored infant activity was independent of the mother's activity. 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.

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 28.8 to 35.8 mg (mean, 31.3 ± 0.6). Taking into account the body weight of each infant, the estimated dose range from 4.00 to 6.41 mg/kg (mean, 5.24 ± 0.2); this is similar to what would be experienced at the breast after the consumption of a 0.3 g/kg dose by the mother.^{5,21}

Method for Measuring Infant Sleep and Activity Rhythms

The actigraph (AMA-32 Ambulatory Monitoring, Ardsley, NY), a self-contained microcomputer that consists of a piezoelectric accelerometer, generates a voltage in proportion to the mechanical deflection of the free end as the actigraph is moved.²² 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 the 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-minute epochs and later analyzed via a computer program developed by Sadeh and colleagues (Ambulatory Monitoring Ltd, Ardsley, NY). The automatic scoring algorithms on which this program is based has been validated previously for sleep–wake discrimination in both infants¹⁶ and adults,¹⁷ and for distinguishing between active versus quiet sleep.²³

The following sleep–wake measures were derived from the activity raw data for each 3.5-hour test session: 1) sleep percent (percentage of total minutes spent in sleep); 2) quiet sleep (total minutes the infant spent in quiet sleep); 3) active sleep (total

minutes the infant spent in active sleep); 4) longest sleep period (length of the longest continuous episode of sleep); 5) latency to fall asleep (number of minutes to first sleep bout); 6) number of sleeping bouts; and 7) mean activity count (average number of zero crossings the piezoelectric beam) during wakefulness. All summary statistics reported in this article are expressed as means \pm SEM, and all *P* values represent two-tailed tests.

RESULTS

Although the infants slept for the same number of times during each test session (paired *t* test (12 *df*) = 0.76; *P* = .46; not significant [NS]) (Table 1), there was, on average, a 25% reduction in the length of time spent sleeping after they consumed the alcohol-flavored milk compared with breast milk alone (paired *t* test (12 *df*) = 2.27; *P* = .04). This reduction was apparently attributable to a shortening in the longest sleeping bout (paired *t* test (12 *df*) = 2.29; *P* = .04) and the amount of time spent in active sleep (paired *t* test (12 *df*) = 3.30; *P* = .006). Although not significant, infants also tended to fall asleep sooner (paired *t* test (11 *df*) = 1.50; *P* = .16; one infant did not sleep during the alcohol test session) and to be more active during wakefulness (paired *t* test (12 *df*) = -1.67 ; *P* = .12) after the ingestion of the alcohol in breast milk compared with breast milk alone.

The decrease in active sleep after alcohol exposure was observed in all but 2 of the 13 infants tested (Fig 1). However, the effect of alcohol was not immediate; ie, there was no significant difference in the amount of time spent in active sleep during the first half of the 3.5-hour testing session (control vs alcohol, 18.2 ± 3.8 vs 17.0 ± 4.2 minutes; paired *t* test (12 *df*) = 0.21; *P* = .84; NS). In contrast, infants spent significantly less time in active sleep during the second half of testing session (ie, 1.75 to 3.5 hours) after exposure to alcohol in breast milk compared with breast milk alone (control vs alcohol, 25.2 ± 5.5 vs 8.6 ± 2.6 minutes; paired *t* test (12 *df*) = 3.14; *P* = .009).

Mothers apparently were unaware of the differences in their infants' behaviors after alcohol exposure; ie, they were as likely to report that they thought their infants consumed the alcohol milk on either test day (χ^2 (1 *df*) = 1.30; *P* > .2; NS). Moreover, there was no significant difference in the number of times the infants breastfed (control vs alcohol, 2.3 ± 0.3 vs 2.6 ± 0.4 ; paired *t* test (12 *df*) = -1.00 ;

TABLE 1. Sleep and Activity Measures During the 3.5 Hours After the Infants' Ingestion of Breast Milk With Alcohol or With Breast Milk Alone

Variable	Type of Milk Ingested by Infant	
	Breast Milk	Alcohol-flavored Breast Milk
Total sleep (min)	78.2 \pm 10.6	56.8 \pm 11.0*
Quiet sleep (min)	34.0 \pm 6.9	31.0 \pm 6.8
Active sleep (min)	44.2 \pm 5.9	25.8 \pm 5.1*
Latency to first sleep bout (min)	50.4 \pm 7.7	34.1 \pm 6.1
Longest sleep bout (min)	56.7 \pm 10.8	34.5 \pm 6.6*
Number of sleeping bouts	2.8 \pm 0.5	2.4 \pm 0.4
Mean activity count during wakefulness	211.9 \pm 6.6	221.9 \pm 6.7

* *P* \leq .05.

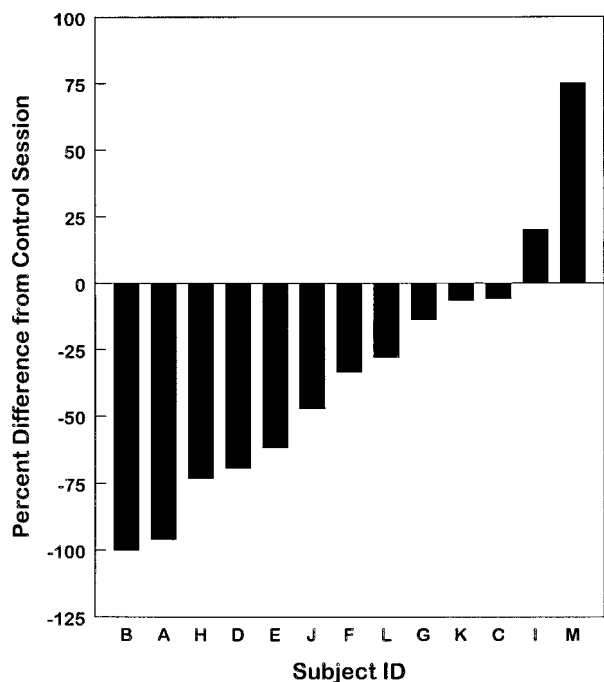


Fig 1. Percent difference in the time each infant spent in active sleep during the 3.5-hour testing session, during which they ingested breast milk flavored with alcohol, compared with time spent in active sleep when they ingested breast milk alone (control condition). The 13 infants are rank-ordered by the amplitude of their responses. Mean percent difference, $-33.8 \pm 13.5\%$; paired t test ($12\ df$) = 3.30; $P = .006$.

$P = .34$; NS) or the amount of milk consumed during these breastfeeds (control vs alcohol, 147.5 ± 19.4 vs 177.6 ± 19.8 mL; paired t test ($12\ df$) = -1.43 ; $P = .18$; NS).

DISCUSSION

The present study expanded on our previous findings⁵ and revealed that acute exposure to alcohol in breast milk altered the infants' sleep-wake patterning; ie, infants tended to fall asleep sooner, but slept for significantly shorter periods of time, during the 3.5 hours after the consumption of breast milk flavored with alcohol compared with consumption of breast milk alone. This reduction apparently was, in part, attributable to a shortening in the amount of time that the infants spent in active sleep (Fig 1), which was evidenced during the latter half of the testing session. Such findings are consistent with those observed previously in healthy adults²⁴⁻²⁶ and other animals²⁷ after the consumption of acute doses of alcohol. Also consistent with previous studies is the finding that infants tended to be more active during wakefulness after consuming the alcohol-flavored milk. Unlike high doses, low doses of alcohol produce a stimulatory effect on locomotor activity in both postweanling²⁸ and adult²⁹ animals.

Several hypotheses, not mutually exclusive, could account for these changes in the infants' behavior after the consumption of alcohol in breast milk. First, the alterations in the infants' behaviors may be in response to the flavoring of the milk. Previous work in our laboratory demonstrated that the mother's ingestion of alcohol, as with many other foods and

beverages, imparts a distinctive change of flavor in breast milk that can be detected by, and perhaps is arousing to, the infant.³⁰ Moreover, animal model research revealed that such sensory experiences can modulate behavior and activity.^{28,31} The reduction in active sleep observed after the ingestion of alcohol in breast milk was not immediate, however.

To determine whether the changes in the infants' sleep behaviors were attributable to the experience with a flavor in breast milk per se, we repeated the study on another group of breastfed infants of similar age. In place of alcohol, nonalcohol-based vanilla was used to examine infants' responses after exposure to a flavor in breast milk on one test day and to breast milk alone on the other.³² Vanilla was chosen because it too is transmitted to breast milk and often is experienced as a sweet odor that shares similar hedonic and flavor properties with low concentrations of ethanol.²⁰ As demonstrated in Figure 2, and in contrast to the infants' response after alcohol exposure (solid circles), there was no significant difference in the amount of time the infants spent in active sleep during the 3.5-hour testing session in which they ingested their mothers' breast milk flavored with vanilla (open circles) compared with breast milk alone. Nor were there significant differences in the number of sleeping bouts, amount of time spent in quiet or total sleep, latency to sleep, longest sleep bout, or activity levels during wakefulness after exposure to the vanilla-flavored milk, thus suggesting that it is not the flavor per se that is responsible for the alterations in sleep-wake patterning after exposure to alcohol in breast milk.

Second, the results observed may be attributable to some changes in the infants' interaction with their mothers. To be sure, sleep, the most frequent state of consciousness for infants, can be influenced by a

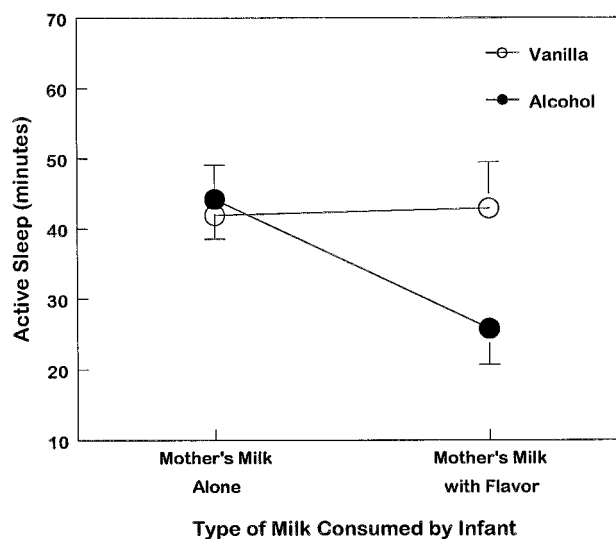


Fig 2. Mean number of minutes the infants spent in active sleep during the 3.5-hour testing session, during which they consumed breast milk alone or breast milk with an added flavor. The solid circles indicate the data from the infants in the present study who were tested with the flavor of alcohol; the open circles indicate data collected from another group of breastfed infants of similar age ($n = 14$) who were tested with nonalcohol-based vanilla flavor (McCormick, Inc, Hunt Valley, MD); $F(1,25\ df) = 5.77$; $P = .02$.

variety of environmental and physiologic factors. However, the present study aimed to control for such factors experimentally. Each of the two test sessions occurred at the same time of day, and testing occurred in a private, quiet room. The infants were placed in the crib or on the carpet as much as possible so that the actigraph-monitored sleep and activity measures were independent of their mothers' activity. The mothers were unaware of the order of testing and, perhaps more importantly, did not identify reliably which day their infants consumed the alcohol-flavored milk. Moreover, there was no difference in the patterning of feeding immediately before and during the two testing days that were separated by an interval of 1 week. Although possible, that the alterations in the patterning of sleep were attributable to changes in the infants' interaction with their mothers seem unlikely. Rather, it appears that exposure to this small amount of alcohol in breast milk had a direct, albeit subtle, effect on the infants.

Because the drug in the nursing infant's blood or urine was not measured, we do not know the amount that was absorbed from the milk. However, the information available on pediatric pharmacokinetics has demonstrated significant differences in the absorption, distribution, metabolism, and excretion of a variety of drugs in infants compared with older children and adults.³³ Some evidence suggests that infants have limited capacity to oxidize ethanol,³⁴ which in turn may render the dose more potent. Animal model studies have revealed that infant rats exhibit a lower alcohol-related metabolic capacity, longer half-life, and in turn higher peak blood alcohol levels,³⁵ and that infant rats are more sensitive to the effects of alcohol on certain cognitive processes^{28,36} compared with older conspecifics.

Despite the disruptions observed in active sleep after exposure to alcohol in their mother's breast milk, it is possible that the infants might later exhibit compensatory increases in active sleep (ie, after the 3.5-hour test session). That they are capable of such compensations is suggested by the observation that infants of mothers who drank heavily throughout pregnancy but were primarily formula-fed after birth spent more time in both quiet and active sleep during the immediate postpartum period.³⁷⁻⁴⁰ Compensatory increases in active sleep also have been reported toward the latter portion of the night after acute consumption of alcohol in nonalcoholic adults^{24,26} and, although tolerance to the sleep-disrupting effects of alcohol occurs within a few nights, compensatory increases in active sleep reoccur when alcohol is discontinued.²⁴

Because the mothers of the infants in the present study drank very little during both pregnancy and lactation, we do not know whether infants who are frequently exposed to alcohol in breast milk would experience continued alterations in sleep-wake patterning. Nevertheless, the finding that acute exposure reduces the time spent in active sleep (but not quiet sleep) may shed light on the aforementioned epidemiologic findings that revealed that the infants who were chronically exposed to alcohol in breast

milk exhibited a slight deficit in motor, but not mental, development, at 1 year of age.¹⁴ Of particular interest is the finding that the degree of abnormality in the electroencephalogram during active sleep at birth was related to subsequent motor, but not mental, development.⁴⁰ Recall that there was no significant difference in the mental development of 1-year-old breastfed infants whose mothers drank daily compared with that for infants whose nursing mothers drank less than one drink per day or those who were formula-fed. Thus, we hypothesize that continued exposure to alcohol in breast milk leads to continued disruption of active sleep, which has been found to be predictive of later motor development in both human infant⁴⁰ and animal model^{41,42} studies.

Although the amount of alcohol ingested in breast milk is a minute fraction of that consumed by the mother, the present study revealed that such exposure may subtly affect the infants' behaviors in the short term. Such findings are consistent with the traditional notion that the infant's behavior can be influenced by the components in breast milk.^{1,2,20} Perhaps one reason why the lore that relates that occasional drinking by the nursing mother can sedate the breastfed infant⁴ has been promulgated is because the infants tended to fall asleep sooner. To be sure, the findings that infants slept less and tended to be more aroused during wakefulness are more consistent with a stimulatory effect of alcohol; however, these effects are subtle and do not address the issue of whether exposure to higher doses of alcohol in breast milk has more pronounced sedative effects on the recipient infant.¹²

The mechanisms underlying these changes in sleep patterning and the long-term impact on development remain to be determined. Unlike pregnant women, nursing women who drink occasionally can limit their infant's exposure to alcohol by timing breastfeeding in relation to their drinking. Contrary to popular beliefs, alcohol is not stored in breast milk, but peaks ~30 minutes to 1 hour after the cessation of drinking and decreases thereafter, much like that found in maternal plasma.^{5,8,9,21} As advocates of breastfeeding, we emphasize the many advantages to both the mother and the infant and encourage health professionals to inform mothers of the scientific information, albeit limited, on the transfer of alcohol to breast milk and its effects on their infant.

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REFERENCES

1. Mennella JA. Mother's milk: a medium for early flavor experiences. *J Hum Lac.* 1995;11:39-45
2. Fildes V. *Breasts, Bottles and Babies: A History of Infant Feeding.* Edinburgh, Scotland: Edinburgh University Press; 1986
3. Adams LM, Davidson M. Present concepts of infant colic. *Pediatr Ann.* 1987;16:817

4. Lawrence RA. *Breastfeeding: A Guide for the Medical Profession*. St Louis, MO: CV Mosby Co; 1989
5. 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
6. Berlin CM Jr. Drugs and chemicals: exposure of the nursing mother. *Clin Pharmacol*. 1989;36:1089-1097
7. Lindmark B. Maternal use of alcohol and breast-fed infants. *N Engl J Med*. 1990;322:338-339. Letter
8. Kesaniemi YA. Ethanol and acetaldehyde in the milk and peripheral blood of lactating women after ethanol administration. *J Obstet Gynaecol Br Commonwealth*. 1974;81:84-86
9. Lawton ME. Alcohol in breast milk. *Aust NZ J Obstet Gynaecol*. 1984;25:71-73
10. American Academy of Pediatrics, Committee on Drugs. The transfer of drugs and other chemicals into human milk. *Pediatrics*. 1994;93:137-150
11. Bisdorf CJW. Alcohol—en nicotinevergiftiging bij zuigelingen. *Maand-schr Kindergeneesk*. 1937;6:332-341
12. Binkiewicz A, Robinson MJ, Senior B. Pseudo-Cushing syndrome caused by alcohol in breast milk. *J Pediatr*. 1978;93:965-967
13. Yamagishi M, Iwasaki T. Acute alcohol intoxication in a two-month-old baby. *J UOEH*. 1987;9:53-59
14. 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
15. Scher A, Epstein R, Sadeh A, Tirosh E, Lavie P. Toddler sleep and temperament: reporting bias or a valid link? A research note. *J Child Psychol Psychiatry*. 1992;33:1249-1254
16. Sadeh A, Lavie P, Scher A, Tirosh E, Epstein R. Actigraphic home monitoring of sleep disturbed and control infants: a new methods for pediatric assessment of sleep-wake patterns. *Pediatrics*. 1991;87:494-499
17. Sadeh A, Alster J, Urbach D, Lavie P. Actigraphically based automatic bedtime sleep-wake scoring. Validity and clinical applications. *J Amb Monitoring*. 1989;2:209-216
18. Sokol RJ, Miller SI, Debanne S, et al. The Cleveland NIAAA prospective alcohol-in-pregnancy study: the first year. *Neurobehav Toxicol Teratol*. 1981;3:203-209
19. Little RE, Worthington-Roberts B, Mann SL, Uhl CN. Test-retest reliability of diet and drinking estimates from pregnancy and post partum. *Am J Epidemiol*. 1984;120:794-797
20. 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. *Alcohol and Alcoholism: Brain and Development*. Hillsdale, NJ: Lawrence Erlbaum Associates. In press
21. Mennella JA, Beauchamp GK. Beer, breast feeding and folklore. *Dev Psychobiol*. 1993;26:409-413
22. Colburn TR, Smith BM, Guarini JJ, Simmons NN. An ambulatory activity monitor with solid state memory. *ISA Trans*. 1976;15:149-154
23. Sadeh A, Acebo C, Seifer R, Aytur S, Carskadon MA. Activity-based assessment of sleep-wake patterns during the 1st year of life. *Inf Behav Dev*. 1995;18:329-337
24. Yules RB, Feedman DX, Chandler KA. The effect of ethyl alcohol on man's electroencephalographic sleep cycle. *Electroencephalogr Clin Neurophysiol*. 1966;20:109-111
25. Williams DL, MacLean AW, Cairns J. Dose-response effects of ethanol on the sleep of young women. *J Stud Alcohol*. 1983;44:515-523
26. Rundell OH, Lester BK, Griffiths WJ, Williams HL. Alcohol and sleep in young adults. *Psychopharmacologia (Berl)*. 1972;26:201-218
27. 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
28. Lopez MF, Spear NE, Molina JC. Ontogenic differences in the expression of olfactory-conditioned versions resulting from a state of acute alcohol intoxication in the rat. *Alcohol*. 1996;13:473-481
29. Pohorecky LA. Biphasic action of ethanol. *Biobehav Rev*. 1977;1:231-240
30. Mennella JA. The human infants' suckling responses to the flavor of alcohol in mother's milk. *Alcohol: Clin Exp Res*. 1997;21:581-585
31. Molina JC, Chotro MG, Dominguez HD. Fetal alcohol learning resulting from alcohol contamination of the prenatal environment. In: Lecanuet J-P, Fifer WP, Krasnegor NA, Smotherman WP, eds. *Fetal Development: A Psychobiological Perspective*. Hillsdale, NJ: Lawrence Erlbaum Associates; 1995:419-438
32. Mennella JA, Gerrish CJ, Hanson K, Groff J. The effects of exposure to alcohol in mother's milk on the infants' sleep and activity levels. *Alcoholism: Clin Exp Res*. 1997;21:A686
33. Milsap RL, Jusko WJ. Pharmacokinetics in the infant. *Environ Health Perspect*. 1994;102:107-110
34. Pikkarainen PH, Raiha NCR. Development of alcohol dehydrogenase activity by the human liver. *Pediatr Res*. 1967;1:165-168
35. Kelly SJ, Bonthuis BS, West JR. Developmental changes in alcohol pharmacokinetics in rats. *Alcohol Clin Exp Res*. 1987;3:281-286
36. Chen W-J, 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
37. 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
38. Rosett HL, Snyder P, Sander LW, et al. Effects of maternal drinking on neonate state regulation. *Dev Med Child Neurol*. 1979;21:464-473
39. Sher 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
40. Ioffe S, Chernick V. Prediction of subsequent motor and mental retardation in newborn infants exposure to alcohol in utero by computerized EEG analysis. *Neuropediatrics*. 1990;21:11-17
41. Mirmiran M, Scholtens J, Van de Poll NE, Ulings HB, Boer GJ. Effects of experimental suppression of active (REM) sleep during early development upon adult brain and behavior in the rat. *Dev Brain Res*. 1983;7:227-286
42. Mirmiran N, Van de Poll NE, Corner MA. Suppression of active sleep by chronic treatment with clomipramine during postnatal development: effects upon adult sleep and behavior in the rat. *Brain Res*. 1981;204:129-146