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Short-Term Effects of Maternal Alcohol Consumption on Lactational Performance

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Previous research demonstrated that breast-feeding infants consumed significantly less milk during the immediate hours after their mothers consumed an acute dose of alcohol when compared with a nonalcoholic beverage. The present study tested the hypothesis that maternal alcohol consumption decreases the amount of milk available to the infant and alters milk composition in the short term. To this aim, 22 lactating women were tested on 2 days separated by 1 week; the women reported that they drank very little during pregnancy, but significantly increased alcohol intake during lactation. Each woman drank a 0.3 g/kg dose of alcohol in orange juice on one testing day and orange juice alone on the other; the order was counterbalanced. Immediately before drinking the beverage (baseline) and 2 hr after (postconsumption), women expressed their milk by using an electric breast pump until no milk had been secreted from either breast for 5 min. Although there was no difference in the energy content of the milk, maternal alcohol consumption slightly, but significantly, reduced the amount of milk produced by the lactating mother. These findings underscore the importance of determining whether and when infants compensate for the reductions in intake experienced at the breast following maternal alcohol consumption and how such changes impact on mother–infant interaction.

Key Words: Alcohol, Lactation, Breast Milk, Nutrition, Development.

DRINKING SMALL quantities of alcohol during lactation is believed to benefit both the mother and her breast-feeding infant.[1] Women in Mexico are encouraged to drink as much as two liters of *pulque* (a low alcoholic beverage made of fermented fruit juice) daily during both pregnancy and lactation,[2] Indochinese women in California drink wine steeped with herbs,[3] whereas the magic elixir in Germany is malt beer.[4] Almost half of the lactating women who participated in a recent study conducted in Philadelphia reported that their health professionals encouraged them to drink alcohol to improve the quality and quantity of their milk or to facilitate milk let-down[5]; this percentage is remarkably similar to that reported in the late 1970s.[6]

The claims that alcohol is a galactagogue are not accompanied by any controlled scientific evidence. In fact, contrary to the lore that alcohol enhances milk intake, but consistent with research in other animals,[7–9] human infants consumed *less* milk during the immediate hours following their mothers' consumption of an acute dose of alcohol.[10,11] The observed decrease in milk intake was not because infants were feeding for shorter periods of time following maternal alcohol consumption.[10,11] Nor was it due to the infants rejecting their mother's milk because the milk's flavor changed after she consumed alcohol.[5]

Although infants consume ~23% less milk during the 4 to 5 hr following their mothers' consumption of alcohol,[10,11] we do not know whether they are consuming less calories because no research, to our knowledge, has focused on the effects of acute ethanol consumption on the composition of human milk. Therefore, the present study tested the hypothesis that maternal alcohol consumption decreases the amount of milk available to the infant and alters milk composition in the short term. Because the composition of milk varies throughout the day and because of individual variation,[12] a within-subjects design that controlled for time of day was implemented. Moreover, mothers used an electric breast pump to express their milk to eliminate the potential confounding variables (e.g., variability in sucking intensity) contributed by the infant.

METHODS

Subjects

Twenty-two lactating women, who had consumed at least one alcoholic beverage during lactation and who had experience using a breast pump, were recruited from ads in local newspapers and from the Women, Infant and Children Centers in Philadelphia. The mothers (9 primiparous, 13 multiparous) ranged in age from 24 to 42 years (mean = 31.9 ± 1.1), and their infants (11 girls, 11 boys) were, on average, 4.6 ± 0.4 months. Mothers were instructed not to alter their breast feeding and/or pumping schedule and to refrain from drinking any alcoholic beverages during the 3 days that preceded each of the two testing days. To encourage compliance, they were asked to record (1) the types of foods and beverages consumed and (2) when they nursed their child or pumped their breasts during these two, 3-day periods. The latter revealed that the infants breast-fed, on average, every 2 hr during the mid-morning and early afternoon hours.

Using a timeline follow-back questionnaire,[13] each woman estimated the number, types, and frequency of alcoholic beverages consumed during pregnancy and lactation. The mothers reported that they drank very little during pregnancy (mean ± SEM = 2.9 ± 0.9/month; range = 0 to 16 drinks/month), but significantly increased alcohol intake during lactation to, on average, 7.9 ± 1.7 alcoholic beverages per month [paired $t(21df) = -3.77$; $p = 0.001$; range = <1 to 24 drinks/month]; these numbers likely underestimate alcohol usage.[14]

All procedures used in this study were approved by the Committee on Studies Involving Human Beings at the University of Pennsylvania, and informed consent was obtained from each woman prior to testing. At the end of the study, we asked each woman to forgo drinking one alcoholic

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Table 1. Effects of Maternal Alcohol Consumption on Lactational Performance at Baseline (11 AM–Noon) and 2 Hr After Consuming Either a 0.3 g/kg Dose of Alcohol in Orange Juice (Alcoholic Beverage) or Orange Juice Alone (Control Beverage)

Time of milk collection: Type of beverage consumed	Baseline (before beverage consumption)		2-hr postbeverage consumption	
	Control	Alcohol	Control	Alcohol
Response measures				
Latency to milk ejection (sec)	34.2 ± 6.9	33.7 ± 6.1	34.0 ± 6.8	37.8 ± 5.7
Milk yield (ml)	95.3 ± 9.0	101.6 ± 7.2	89.5 ± 5.8	79.4 ± 5.3*
Length of collection period (min)	17.1 ± 1.2	16.4 ± 0.8	15.7 ± 0.9	15.5 ± 0.8
Caloric content of milk (kcal/dl)	95.5 ± 2.5	95.8 ± 3.3	87.1 ± 2.2	87.5 ± 2.7
Fat content (g/liter)				
Foremilk	8.8 ± 0.5	8.5 ± 0.4	7.6 ± 0.4	7.7 ± 0.3
Hindmilk	11.8 ± 0.7	12.4 ± 0.8	10.5 ± 0.5	10.1 ± 0.6

* $p < 0.05$, when compared with control condition (2 hr postconsumption).

Table 2. Statistical Evaluation of Results*

	Time of milk collection (baseline, 2 hr)		Type of beverage consumed (control, alcohol)		Interaction effects: time of collection × beverage type	
	F	*p*	*F*	*p*	*F*	*p*
Response measures						
Latency to milk ejection (sec)	0.23	0.64	0.04	0.84	0.25	0.62
Milk yield (ml)	8.34	0.009	0.31	0.58	4.60	0.04
Length of collection period (min)	4.47	0.05	0.51	0.48	0.17	0.69
Caloric content of milk (kcal/dl)	9.30	0.006	0.02	0.90	0.00	0.97
Fat content (g/liter)						
Foremilk	11.57	0.003	0.03	0.87	0.90	0.35
Hindmilk	19.36	0.0003	0.029	0.87	2.25	0.15

* A repeated-measures analysis of variance was conducted for each measure, with time of collection (baseline, 2 hr postconsumption of the beverage) and type of beverage consumed during the testing session (control beverage: orange juice, alcoholic beverage: alcohol in orange juice) as the repeated factors.

beverage in the near future so that she would not be exposed to additional alcohol as a result of her participation in this study.

Procedures

Each woman was tested on 2 days separated by 1 week (±2 days). The mother arrived at the Monell Chemical Senses Center at ~9:30 AM, having last fed her infant at approximately the same time on each testing day. After acclimatization to the room and personnel, each mother expressed milk from both breasts simultaneously by using an electric breast pump with a special double-pump adaptor (Medela, Crystal Lake, IL); the setting for the intensity of pumping remained constant throughout the session. Pumping ceased when no milk had been secreted from either breast for 5 min. This collection was to ensure standardization of and familiarization with the testing procedures and appropriate baseline measures. Moreover, mothers used this and other expressed milk to feed their infants during the next 4 hr.

The entire collection procedure was repeated 2 hr later (Baseline Collection), after which the mother drank either a 0.3 g/kg dose of alcohol in orange juice or an equal volume of orange juice alone within a 15-min period.[10,11] Half of the women drank the alcohol in orange juice during the first testing day and the orange juice alone during the second testing day; the order was reversed for the remaining women. The mothers again expressed milk 2 hr after consuming the beverage (Postconsumption Collection). No effect of order was observed for any of the variables we investigated.

Response Measures

For both the baseline and postconsumption collection periods, we recorded the amount of time it took for the first droplet of milk to be ejected (*latency to eject*), the volume of milk expressed from each breast within each 5-min period (*milk yield*), and the total length of the collection period. Because our primary goal was to determine the amount and type of calories available to the infant, the milk from both breasts was pooled. From this pooled sample of milk, we determined, in triplicate, its caloric content. However, because of the extreme variability in the fat content of human milk within a feed,[15] the fat content was determined on both foremilk and hindmilk samples prior to pooling (see herein). The following summarizes each of these methodologies.

Caloric Content. The energy content (kcal/g) of human milk was determined directly in an adiabatic bomb calorimeter (Parr model 1341, Moline, IL) on each pooled sample. That is, the heats of combustion of a weighed amount (~0.2 g) of the milk sample was combusted with a known amount (0.8 ml) of mineral oil using the methods described by Butte and colleagues[16] and Miller and Payne.[17]

Fat Content. The fat concentration of both the first 5-min (foremilk) and last 5-min (hindmilk) sampling for each collection was determined using the creamatocrit technique.[15] The milk was immediately drawn into three microcapillary tubes and sealed with Critoseal, after which the tubes were spun for 10 min in a microcapillary centrifuge. The height of the cream layer was measured and then converted to fat (grams/liter).

Statistical Analyses

A repeated-measures analysis of variance was conducted for each measure with time of collection (baseline, 2-hr postconsumption) and type of beverage consumed (orange juice, alcohol in orange juice) as the repeated factors. After significant interaction effects, paired *t* tests were performed to assess differences. All summary statistics are expressed as mean ± SEM, and all *p* values represent two-tailed tests.

RESULTS

Table 1 presents the means (±SEM) and Table 2 presents the results of the statistical analyses on each of the measures evaluated. A significant interaction between the time of collection (baseline, 2-hr postconsumption) and the type of beverage consumed on the testing day (control, alcohol) was observed for the amount of milk produced by

the mother. Further analyses revealed that there was no significant difference in the amount of milk pumped at baseline [paired $t(21df) = -1.06$; $p = 0.30$], but mothers pumped significantly less milk 2 hr after consumption of the alcoholic beverage when compared with the amount pumped 2 hr after consuming the control beverage [paired $t(21df) = 2.45$; $p = 0.02$; see table 1]. Mothers pumped, on average, 9.3% (±4.1) less milk during the 2 hr following maternal alcohol consumption. This decrease in milk production tended to be apparent during the first 5 min of pumping during the 2-hr postconsumption collection period [control vs. alcohol: 53.6 ± 5.2 vs. 50.1 ± ml; paired $t(21df) = 1.54$; $p = 0.13$)].

As expected, there was a significant effect of the time of collection on the fat and caloric content of the milk. Further analyses revealed that on both testing days, mothers produced a higher caloric milk [control day: paired $t(21df) = 2.59$; $p = 0.02$; alcohol day: paired $t(21df) = 2.32$; $p = 0.03$] during the baseline collection that occurred at midday when compared with the 2-hr postconsumption collection that occurred at 2 PM. However, there was no significant effect of type of beverage consumed, or significant (time of collection × type of beverage consumed) interactions for any of these measures.

DISCUSSION

Human milk is not a uniform bodily fluid but one of changing composition. As demonstrated in the present study, not only is there a great deal of variation within a feed, but its composition changes as a function of the time of day and individual variation, with the most variable macronutrient being its fat content.[18,19] We found no effects of acute ethanol consumption during lactation on the energy content of the milk, although animal model studies demonstrated that *chronic* ethanol consumption during lactation resulted in significant reductions in milk yield,[20] as well as an altered fatty acid (i.e., decrease in phosphatidyl serine content) profile of individual phospholipid when compared with control dams.[21] Whether such effects were due to a direct consequence of prolonged drinking or malnutrition is not known, however. Nor do we know whether chronic alcohol consumption in humans has similar effects on the fatty acid profile of human milk, because few of the women in present study reported drinking frequently.

The present study also demonstrated that mothers produced a slight, but significant, decrease in milk yield 2 hr after consuming an alcoholic beverage when compared with a nonalcoholic beverage. Because their infants were feeding approximately every 2 hr, this 9% decrease represents the amount of milk available to the infant during *one* breast feed. Recall that previous research, which focused on milk intake at the breast, demonstrated that infants consumed ~23% less milk during the 4 to 5 hr (or 2 to 3 breastfeeds) that followed their mothers' consumption of an alcoholic beverage.[10,11] However, it is important to note that, under normal breastfeeding conditions, milk can usually be expressed from the breast after a breastfeed, suggesting that the amount of milk available in the breasts is not always an important determinant of the amount of milk withdrawn by the infant.[19]

That there was no effect of an acute dose of alcohol (0.3 g/kg) on the latency to eject milk is supported by earlier work conducted by in the late 1960s, in which intramammary and intrauterine pressure changes in response to infant's suckling at the breast were measured as an indicator of the milk-ejection reflex. That is, pressure changes were not affected in peripartum women who received alcohol dosages <1 g/kg of body weight, whereas partial or complete blockage of the reflex was observed in those who received dosages >1 g/kg.[22–24] Whether the consumption of alcohol at dosages similar to that used in the present study could affect the endocrine milieu of the mother, and in turn, the amount of milk secreted, remains unknown.

To our knowledge, the only research on the effects of alcohol on the endocrine milieu of the lactating mother are limited to animal model studies that revealed that acute alcohol administration did not affect basal prolactin levels, but significantly inhibited suckling-induced prolactin release in lactating rats, as well as milk intake by their pups.[25–28] Subramanian and colleagues[29] hypothesized that alcohol inhibits suckling-induced prolactin release by either disrupting the transmission of neural impulses emanating from the nipples to the central nervous system or acting directly on the hypothalamus or higher centers that modulate prolactin releasing and inhibiting factors. Recent data suggest that alcohol may be lowering serotonergic activity in hypothalamic areas (e.g., arcuate, dorsomedial, paraventricular, periventricular, and supraoptic) important for prolactin release.[29]

In summary, this study has shown that maternal alcohol consumption slightly, but significantly, reduced the amount of milk produced by lactating women further supporting the hypothesis that alcohol may be directly affecting the mothers' lactational performance. The effects are subtle, but the methodologies described herein have been found to be sufficiently sensitive to reveal differential responses in lactational performance as a function of maternal alcohol consumption. Moreover, the finding that there was no significant difference in the caloric content of the milk further demonstrates the importance of determining whether and when infants compensate for such reductions in intake and how such changes impact on mother–infant interaction.

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