Published in final edited form as:
J Clin Endocrinol Metab. 2005 April ; 90(4): 1979–1985.

Acute Alcohol Consumption Disrupts the Hormonal Milieu of Lactating Women

Julie A. Mennella, **M. Yanina Pepino**, and **Karen L. Teff**
Monell Chemical Senses Center (J.A.M., M.Y.P., K.L.T.), Philadelphia, Pennsylvania 19104-3308

Department of Medicine (K.L.T.), Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania 19104

Abstract

Despite the lack of scientific evidence to support the claim that alcohol is a galactagogue, lactating women have been advised to drink alcohol as an aid to lactation for centuries. To test the hypothesis that alcohol consumption affects the hormonal response in lactating women, we conducted a within-subjects design study in which 17 women consumed a 0.4 g/kg dose of alcohol in orange juice during one test session and an equal volume of orange juice during the other. Changes in plasma prolactin, oxytocin, and cortisol levels during and after breast stimulation, lactational performance, and mood states were compared under the two experimental conditions. Oxytocin levels significantly decreased, whereas prolactin levels and measures of sedation, dysphoria, and drunkenness significantly increased, during the immediate hours after alcohol consumption. Changes in oxytocin were related to measures of lactational performance such as milk yield and ejection latencies, whereas changes in prolactin were related to self-reported measures of drunkenness. Although alcohol consumption resulted in significantly higher cortisol when compared with the control condition, cortisol levels were not significantly correlated with any of the indices of lactational performance or self-reported drug effects. Moreover, cortisol levels steadily decreased on the control day, indicating that the procedures were not stressful to the subjects. In conclusion, recommending alcohol as an aid to lactation may be counterproductive. In the short term, mothers may be more relaxed, but the

Correspondence to: Julie A. Mennella.

Address all correspondence and requests for reprints to: Julie A. Mennella, Ph.D., 3500 Market Street, Philadelphia, Pennsylvania 19104-3308. E-mail: mennella@monell.org..

This work was supported by the National Institutes of Health Grant R01AA09523, Diabetes Research Center Grant RR00040, National Institute of Diabetes and Digestive and Kidney Diseases Grant 19525, and the Office of Research on Women′s Health.

Present address for K.L.T.: National Institute of Diabetes, Digestive and Kidney Disease, Bethesda, Maryland.

Abbreviations:

ARCI Addiction Research Center Inventory

AUC area under the curve

BAC blood alcohol concentration(s)

LSD Lysgeric

PCAG Pentobarbital-Chlorpromazine-Alcohol Group

JCEM is published monthly by The Endocrine Society (http://www.endo-society.org), the foremost professional society serving the endocrine community.

hormonal milieu underlying lactational performance is disrupted, and, in turn, the infant's milk supply is diminished.

THE TRADITIONAL WISDOM of many cultures relates that women can optimize the quality and quantity of their milk to meet the needs of their infants through diet and psychological well-being. Each culture claims some milk-producing (galactogenic) substances, and many cultures claim alcohol to be such a substance (1). Such beliefs were so ingrained in American tradition that, in 1895, Anheuser-Busch Company, a major U.S. brewery, produced Malt-Nutrine, a low-alcoholic beer that was sold exclusively in drugstores and prescribed by physicians as a tonic for pregnant and lactating women (2). Even in more modern times, a popular book for nursing mothers hailed the virtues of alcohol as a galactagogue, claiming ". . . this is one time in life when the therapeutic qualities of alcohol are a blessing " (3).

Such claims have not gone unchallenged. In 1987, the *Journal of the American Medical Association* published a letter from a physician asking whether there was any scientific basis for prescribing a daily beer to lactating women (4). The scientific basis, it was declared (5), can be found in the finding that the consumption of beer, unlike other alcoholic beverages, increases serum prolactin (5,6). There are several problems with this conclusion, however.

First, the subjects in these research studies were men and nonlactating women. No study to date had examined the effects of alcohol consumption on the hormonal milieu of lactating women, additionally highlighting the lack of evidence-based practice related to recommendations regarding alcohol consumption during lactation. There has been considerable research in animal models (for review, see Ref. 7), however. Although the vast majority of these studies reported that ethanol administration decreased suckling-induced prolactin, the most recent study, which extended the observation period (8), actually demonstrated significant suckling-induced increases in prolactin after ethanol administration. These latter findings led the authors to conclude that oxytocin, rather than prolactin, may be the primary avenue by which alcohol induces growth retardation during lactation.

Second, the rise in prolactin levels after alcohol consumption was observed after the consumption of different types of alcoholic beverages (9,10) and was not specific to beer consumption, as the folklore suggested (2,4,5). Moreover, if alcohol does indeed increase prolactin levels in maternal circulation, it is not apparent whether such increases affect lactational performance. Although prolactin appears to be essential for the initiation of lactation and its maintenance in the long term (11), no clear temporal correlation exists between plasma prolactin levels and milk yield of a particular breastfeed in humans (12).

Third, it is perplexing that one would argue that alcohol enhances lactational performance when this same drug, at similar or slightly higher doses, was used in the not-so-distant past to treat premature labor (13,14). The efficacy of alcohol in partially blocking uterine contractions during labor is due, in part, to its inhibition of oxytocin (14), a hormone that is also involved in the contraction of myoepithelial cells surrounding the alveoli, which, in turn, causes the ejection of milk from the mammary gland during lactation (15).

Fourth, research conducted during the past decade refutes the lore that alcohol is a galactagogue. Rather, lactating mothers produced less milk without changes in the caloric content of their milk (16), and, in turn, infants consumed less breast milk and less calories during the immediate hours after maternal consumption of beer as well as other types of alcoholic beverages (17,18). The fact that infants sucked more during the beginning of the breastfeed (17) suggested that they were having difficulty in obtaining milk from the breast because infants suck at faster rates when milk flow is lower (19).

The present study tested the hypothesis that the alcohol-induced depression in milk production in lactating women was due to disruptions in the hormonal milieu. Oxytocin and prolactin responses were evaluated when lactating women consumed a moderate dose of alcohol, one that was equivalent to one to two drinks and represents the average amount of alcohol lactating women reported consuming during a drinking occasion (20).

Subjects and Methods

Subjects

Seventeen nonsmoking, healthy lactating women (six primiparous and 11 multiparous), who were exclusively nursing infants between the ages of 2 and 4 months, were recruited from ads in local newspapers and newsletters. One additional woman began testing but was excluded because of procedural difficulties. During initial screening, women were excluded if they were lifetime alcohol abstainers, on any medication including oral contraceptives, or had resumed menstruation, because there is some suggestion that both basal and peak prolactin levels are lower in such women (21). All procedures were approved by the Office of Regulatory Affairs at the University of Pennsylvania, and each subject gave informed written consent before testing.

The women (10 Caucasian, five African American, one Asian, and one from another ethnic group) were, on average, 31.9 ± 1.2 yr of age, with a mean body mass index of 26.4 ± 1.1 kg/m^2. They reported that alcohol intake was low during pregnancy (mean = 0.2 ± 0.1 standard drinks per month) but significantly increased to, on average, 1.5 ± 0.6 drinks per month during lactation [paired *t*-test (16df) = -2.14; $P = 0.048$]. These numbers likely underestimate alcohol usage (22).

Procedures

A within-subjects design study that controlled for time of day was employed because milk composition and hormonal responses vary throughout the day. Using methodologies developed for the study of neurally mediated hormonal responses in humans (23), women were tested at the General Clinical Research Center (GCRC) at the University of Pennsylvania on 2 d separated by 1 wk (±2 d). After abstaining from alcohol for at least 3 d, all subjects arrived at the GCRC at 0800 h (±30 min) after an overnight fast and remained fasted during the entire testing procedures, because prolactin levels can be potentiated by certain gastrointestinal hormones and high blood glucose levels (24). Mothers were not allowed to watch television, sleep, or talk, as well as read about food or infants throughout the entire testing session because these behaviors may affect the hormones under study. Instead, they were able to read magazines or novels or to converse on other topics. Moreover, infants were not present because the mere sound, sight, or smell of the baby often stimulates milk let-down or leaking (25). Breast stimulation was provided by an electric breast pump because prior work revealed that infants′ sucking intensity changes when their mothers′ milk contains alcohol (20).

Approximately 30 min after arrival, an iv line was inserted into the antecubital vein of an arm. Because prolactin is very stress labile and rises during the first half-hour after a needle prick (26), subjects acclimated in a private testing room for 45 min. After acclimatization, blood samples were obtained at fixed intervals (-40, -25, and -10 min) before drinking a 0.4 g/kg dose of alcohol in orange juice (15% vol/vol) on one testing day (alcohol condition) and an equal volume of orange juice on the other day (control condition). During both conditions, 3 ml of alcohol were pipetted onto the surface of the cup to serve as a smell and flavor mask (27). The order of testing was randomized between subjects. The beverage was aliquoted into two equal volumes, and each aliquot was consumed within consecutive 5-min periods.

As shown in Table 1, approximately 35 min after subjects started drinking the beverage, blood samples were again taken every 2 min for 16 min before and during stimulation of alternating breasts (35, 37, 39, 41, 43, 45, 47, 49, and 51 min after consumption of beverage) with an electric breast pump (Medela, Crystal Lake, IL), and then every 15 min without breast stimulation for the next 90 min (65, 80, 95, 110, 125, and 140 min). Each sample collection involved the removal of 1 ml of blood to clear the catheter tubing, followed by a 5-ml collection into Vacutainer tubes containing EDTA. Samples were kept on ice for no longer than 1 h and were centrifuged, separated into aliquots, and stored at -70 C for later assay. The latency (in seconds) to eject the first droplet of milk was recorded, and the total amount of milk pumped (in milliliters) during this 16-min period was measured. Nurses were blind to the conditions of the experiment.

Blood alcohol concentrations (BAC; g/liter) were estimated by having subjects breathe into an Alco-Sensor III (Intoximeters, Inc., St. Louis, MO) throughout the test sessions (Table 1). The rationale for the estimation of BAC from "breath alcohol" measurements was based in the simplicity, accuracy, and reliability of the method, which has become a standard procedure in alcohol pharmacokinetic studies (28,29). The Alco-Sensor III device provides readings of BAC levels by assuming a blood to breath ratio of 2100:1. Previous research established that the correlation between ethanol levels measured in blood samples and those measured in exhaled alcohol concentration breath samples was better than 0.98 for both the absorption and elimination phases (30). Subjects also completed the Addiction Research Center Inventory (ARCI) throughout each test session to assess various measures of self-reported drug effects (27). This questionnaire consists of a number of scales including the Morphine Benzedrine Group scale, which measures drug-induced euphoria; the Pentobarbital-Chlorpromazine-Alcohol Group (PCAG) scale, which measures sedation; the Lysgeric (LSD) scale, which measures dysphoric and somatic effects; the Benzedrine Group and Amphetamine scale, which measures stimulant-like effects; and the Drunk Scale, which measures drunkenness.

Hormone assays

Plasma samples were measured in duplicate by double-antibody RIAs for oxytocin and cortisol and by immunoradiometric assay for prolactin. Standards were run with each assay. All samples from a given subject from both days (alcohol and control) of testing were run within the same assay to reduce interassay variability. Cortisol levels were monitored on the control day to ensure that alterations in hormonal responses were not related to the stress of the procedures (10). Intraassay variation was 2.8, 3.0, and 1.3%, and interassay variation was 1.9, 8.9, and 10.2% for oxytocin, prolactin, and cortisol, respectively. All assays were performed by the Diabetes Research Center of the University of Pennsylvania.

Oxytocin was assayed without extraction by using a competitive RIA, with materials supplied by Phoenix Pharmaceuticals, Inc. (Belmont, CA). The antiserum cross-reactivity with arginine vasopressin, GH, α-atrial natriuretic peptide (1-28), methionine-enkephalin, GH-releasing factor, somastatin, TRH, vasoactive intestinal peptide, and pituitary adenylate cyclase-activating polypeptide 27-NH_2 is 0%. The minimal detectable concentration was 10 pg/ml (8 pmol/liter).

Prolactin was assayed by a direct, two-site immunoradiometric assay without extraction, using materials supplied by ICN Diagnostics (Costa Mesa, CA). The antiserum cross-reactivity is less than 0.01% for human chorionic gonadotropin, TSH, LH, and FSH. The minimal detectable concentration was 2.5 ng/ml (108.8 pmol/liter).

Cortisol was measured without extraction by a competitive double-antibody RIA kit from ICN Diagnostics. The antiserum cross-reacts 12.3% with 11-deoxycortisol, 5.5% with corticosterone, and less than 2.7% with all other steroids tested.

Data analyses

Separate repeated measures mixed ANOVA were conducted to determine whether there were significant differences in prolactin, oxytocin, cortisol, and BAC levels, as well as various measures of self-reported drug effects with experimental condition (alcohol and control) and time as the within-subjects factors. When significant, *post hoc* Fisher least significant difference analyses were conducted. Because there were no significant differences in the basal values for oxytocin [F(2,32df) = 2.17; $P = 0.13$] and prolactin [F(2,32df) = 0.50; $P = 0.61$], we calculated changes in prolactin and oxytocin from respective baseline value (mean of three baseline samples) for each subject. There was a significant effect of time on cortisol baseline samples [F(2,32df) = 60.77; $P < 0.0001$]. Therefore, the last sample (t =-10 min) was used as the baseline value. We then determined the peak value for each hormone when compared with baseline and calculated the area under the curve (AUC) values by using a point-to-point method (OriginLab Corporation, Northampton, MA) from baseline to the end of the test session (t = 140 min). The areas for each hormone and for each subject were calculated independently. Paired *t*-tests were used to compare the peak value of each hormone and the AUC between experimental conditions, respectively. The critical value for significance was $P < 0.05$, and all *P* values represent two-tailed tests.

Results

Hormonal responses and lactational performance

Oxytocin. There was a significant interaction between condition and time on oxytocin levels [F(15,240df) = 1.83; $P = 0.03$]. As shown in Fig. 1, oxytocin levels were significantly depressed during and after breast stimulation on the day women consumed alcohol when compared with the control day. Likewise, the oxytocin AUCs were, on average, 78% (±26.6) smaller during the test session in which women consumed alcohol when compared with control [paired t(16df) = -3.22; $P = 0.005$].

Although there were no significant correlations with oxytocin AUC during breast stimulation and milk ejection latency (Table 2), there was a significant correlation between oxytocin levels during the initial minutes of breast stimulation (t = 35-37 min) and the milk ejection latency on the day women consumed alcohol [r(17df) = -0.48, $P = 0.05$; and r(17df) =-0.55, $P = 0.02$, respectively]. That is, the lower the levels of oxytocin, the longer their latency to eject milk. Peak oxytocin levels on the day women consumed alcohol did not significantly differ from peak levels of the control day [paired t(16df) = -1.10; $P = 0.29$]. The individual differences in oxytocin response to breast stimulation were preserved such that oxytocin AUCs on the control day were significantly correlated with AUCs on the alcohol day (Table 2).

Twelve of the 17 women produced less oxytocin during breast stimulation on the alcohol day when compared with the control day ($P < 0.05$). These women also had lower milk yields during the 16 min of pumping when compared with the remaining women [F(1,15df) = 9.35; $P = 0.008$]. They produced, on average, 13 ± 7% less milk during these 16 min of pumping (control *vs.* alcohol, 131 ± 10 *vs.* 113 ± 11 ml). There were no significant relationships between the oxytocin AUCs or oxytocin levels on either the control or alcohol day and any of the self-reported measured indices of drug effects (all *P* values >0.10).

Prolactin. There was a significant interaction between condition and time on prolactin plasma levels [F(15,240df) = 3.31; $P < 0.001$]. As shown in Fig. 1, in contrast to that observed for oxytocin, alcohol significantly magnified the prolactin response both during and after breast stimulation, and peak prolactin levels were significantly higher on the day women consumed alcohol when compared with the control day [paired t(16df) = 2.52; $P = 0.02$]. The AUCs significantly increased, on average, by 336% (±222) during the alcohol session [paired t(16df)

= 3.52; $P = 0.003$]. This enhanced response was observed in 76% of the women tested ($P = 0.02$).

Although there were no significant relationships on the control day between prolactin levels and milk ejection latency or amount of milk expressed (all P values > 0.10), prolactin AUCs during breast stimulation were significantly correlated with milk ejection latencies on the day women consumed alcohol (Table 2). The higher the prolactin levels, the longer the milk ejection latency. However, unlike oxytocin, there was no significant correlation between prolactin AUCs during breast stimulation on the control day and prolactin AUCs on the alcohol day (Table 2). Ratings of drunkenness, as measured by the ARCI, were also significantly correlated with relative increases in prolactin levels when BAC levels were peaking [t = 65 min; r(17df) = 0.60; $P = 0.01$].

Cortisol. Although the repeated measure mixed ANOVA analysis revealed only a tendency for an interaction effect between condition and time for cortisol levels [F(15,240df) = 1.63; $P = 0.07$], there were significant effects of condition [F(1,16df) = 5.91; $P = 0.03$] and time [F(15,240df) = 4.39; $P < 0.001$] on cortisol levels. As shown in Fig. 1, alcohol consumption resulted in significantly higher cortisol when compared with the control condition. Likewise, peak cortisol levels were significantly higher on the day women consumed alcohol [paired t(16df) = 2.17; $P = 0.05$]. However, there was no significant difference in cortisol AUCs between the two conditions [paired t(16df) = 0.94; $P = 0.36$]. Cortisol AUCs were not significantly correlated with prolactin [control day: r(17df) = 0.29, $P = 0.26$; alcohol day: r(17df) = -0.05, $P = 0.84$] or oxytocin AUCs [control day: r(17df) = 0.41, $P = 0.10$; alcohol day: r(17df) = -0.10, $P = 0.72$]. Neither the cortisol AUCs nor cortisol levels on either the control or alcohol day significantly correlated with any of the indices of lactational performance (*i.e.* milk ejection latency, amount of milk expressed) or self-reported drug effects (all P values > 0.10). Figure 1 also shows that cortisol levels steadily decreased on the control day, thus suggesting that the procedures were not stressful to the subjects.

Ethanol pharmacokinetics and self-reported drug effects

BAC peaked approximately 43-51 min after alcohol consumption and decreased thereafter. As shown in Fig. 2, feelings of sedation [F(1,16df) = 39.19; $P < 0.0001$], dysphoria [F(1,16df) = 10.94; $P < 0.005$], and drunkenness [F(1,16df) = 37.95; $P < 0.0001$] significantly increased during the testing session in which women consumed the alcoholic beverage. These changes in self-reported drug effects paralleled the changing blood alcohol levels.

Discussion

Moderate alcohol consumption disrupted the two key hormones underlying lactational performance. During the immediate hours after alcohol consumption, oxytocin levels significantly decreased, whereas prolactin levels significantly increased both during and after breast stimulation. The magnitude and persistence of the hormonal response in lactating women is more robust when compared with men and nonlactating women (5,6,9,10), further highlighting the dynamics of the system under study. The diminished oxytocin response was significantly related to decreases in milk yield and milk ejection. These latter findings suggest that such changes in hormonal responses mediate the diminished milk production by lactating women (16) and disruption in their infants′ suckling behaviors and milk intake observed in prior research (17,18,31), and further dispute the lore that alcohol is a galactagogue.

In contrast to the response observed for oxytocin, prolactin levels significantly increased after alcohol consumption both during and after periods of breast stimulation. The alcohol-induced increases in prolactin were related to self-reported perceptions of drunkenness. Women also reported increased feelings of sedation and dysphoria during the immediate hours after alcohol

consumption. Because sleep deprivation increases feelings of sedation and dysphoria (32), we hypothesize that sleep deprivation, which is common among mothers of young infants, contributed to the increased feelings of sedation and dysphoria observed on the day lactating women consumed alcohol, as discussed herein. Although prolactin levels during breast stimulation were related to milk ejection latency on the day women consumed alcohol, it should be emphasized that no relationships were observed between prolactin levels or AUCs and the amount of milk produced on either test day. This is consistent with prior research revealing that although prolactin appears to be essential for the initiation of lactation and its maintenance in the long term (11), no clear temporal correlation exists between plasma prolactin levels and milk yield of a particular breastfeed in humans (12). It remains to be determined whether the relationship between alcohol-induced changes in prolactin and milk ejection latency was a spurious correlation and secondary to the effect of alcohol on other mediating factors underlying ejection.

Cortisol levels were also increased during the test session in which women consumed the alcoholic beverage, a finding that is consistent with research from animal models (33) and some human studies (34). However, such changes in cortisol were not related to changes in oxytocin or prolactin, measures of lactational performance, or mood states.

The production, secretion, and ejection of milk are the result of highly synchronized endocrine and neuroendocrine processes, which are governed, in part, by the frequency and intensity of the infants′ sucking. Breast stimulation resulted in transient release of both oxytocin and prolactin to levels previously observed by other researchers (35). Although these two key hormones usually behave in tandem under normal conditions, alcohol consumption resulted in differential and divergent responses. We hypothesize that alcohol acts at the central nervous system level through a general depression or by inhibiting synaptic transmission of afferent impulses to the hypothalamus. Such depression or inhibition would decrease oxytocin levels (36), but, because projections from the hypothalamus exert an inhibitory control of prolactin, prolactin levels would increase (37). Whether the enhanced prolactin response is also due to alcohol′s simulation of extrapituitary tissues such as the mammary glands (38), which are capable of producing prolactin, is not yet known. Animal studies suggest that alcohol, directly or indirectly via estrogens, may elevate prolactin by stimulating activity of lactotropes in the adenohypophysis (38).

Recent studies indicate that one fourth of the women surveyed reported that they were encouraged by health professionals to drink once they began lactating (1,39). Advice ranged from the recommendation that drinking alcohol shortly before nursing will facilitate let-down and milk production to the belief that by drinking such milk, the infant will relax, become less "colicky," and obtain warmth. Some health professionals promote moderate drinking (1,39), whereas others caution that extremely high doses (≥1.0 g/kg) inhibit the milk ejection reflex (40). The present findings, which employed more sensitive measures and controls than research conducted in the 1960-1970s (15), revealed that lower doses of alcohol have similar effects on hormonal milieu and lactational performance.

Several explanations, not mutually exclusive, may shed light on why the folklore that alcohol consumption enhances lactational performance has persisted for centuries. First, because difficulties with lactational performance are often attributed to stress, alcohol is then prescribed as an aid to lactation because of its anxiolytic and sedative properties. The present study revealed that relatively low BACs produce slight, but significant, alterations in feelings of drunkenness, dysphoria, and sedation. However, paralleling these mood changes are disruptions in the hormonal milieu that may impair lactational performance. Second, the lactating mother does not have an immediate means of assessing milk yield or intake. Although breast-fed infants consumed, on average, 20% less milk after mothers′ consumption of the

alcohol (17,31), mothers were apparently unaware of this difference (31). Because milk intake and the rate of synthesis of human milk vary from feed to feed, a difference of this magnitude may be difficult for women to perceive, thus making them particularly vulnerable to such folklore. Third, mothers may be noticing changes in their infants′ behaviors that occur several hours after and as a consequence of drinking (41). Fourth, because prolactin levels are correlated with the lactating mother′s perception of the fullness in the breasts (12), alcohol-induced increases in prolactin may lead mothers to feel that they have more milk, despite the alcohol-induced decreases in milk yield (31). Such perceptions may explain why the folklore that alcohol consumption enhances lactational performance has persisted for centuries.

In conclusion, whereas folklore has perpetuated the belief that alcohol is an aid to lactation and lactating women have sometimes been encouraged to drink low or moderate doses of alcohol as a way to increase milk production (1,39), the research discussed herein indicates that alcohol consumption disrupts the two key hormones involved in lactation in the short term; prolactin levels are enhanced, but oxytocin levels are attenuated during breast stimulation. The long-term consequences of such disruptions on lactational performance and women′s health (42, 43), in general, remain unknown.

Acknowledgments

We acknowledge the expert technical assistance of Ms. A. Lorraine Norfleet, M.H.A., B.S.N., B.S., R.N.; the nurses of the General Research Center at The University of Pennsylvania; and Dr. Heather Collins at the Diabetes Research Center, University of Pennsylvania.

References

1. Mennella, JA. Alcohol and lactation: the folklore *versus* the science. In: Auerbach, KG., editor. Current issues in clinical lactation. Jones and Bartlett Publishers; Boston, MA: 2002. p. 3-10.
2. Krebs, R. Making friends is our business—100 years of Anheuser-Busch. Anheuser-Busch Inc; St. Louis, MO: 1953.
3. Pryor, K. Nursing your baby. Harper and Row Publishers; New York: 1963. p. 190
4. Blume S, Auerbach KG, Schreiber JR, Falkner F. Beer and the breast-feeding mom. JAMA 1987;258:2126.
5. Grossman ER. Beer, breast-feeding and the wisdom of old wives. JAMA 1988;259:1016. [PubMed: 3339797]
6. De Rosa G, Corsello SM, Ruffilli MP, Della Casa S, Pasargiklian E. Prolactin secretion after beer. Lancet 1981;2:934. [PubMed: 6117712]
7. Subramanian, MG. Effects of ethanol on lactation. In: Abel, EL., editor. Fetal alcohol syndrome: from mechanism to prevention. CRC Press Inc.; Boca Raton, FL: 1996. p. 237-247.
8. Heil SH, Subramanian MG. Chronic alcohol exposure: extended observations. Alcohol 2000;21:127–132. [PubMed: 10963935]
9. Mendelson JH, Mello NK, Ellingboe J. Acute alcohol intake and pituitary gonadal hormones in normal human females. J Pharmacol Exp Ther 1981;218:23–26. [PubMed: 7241380]
10. Soyka M, Gorig E, Naber D. Serum prolactin increase induced by ethanol—a dose-dependent effect not related to stress. Psychoneuroendocrinology 1991;16:441–446. [PubMed: 1805295]
11. Howie PW, McNeilly AS, McArdle T, Smart L, Houston M. The relationship between suckling-induced prolactin response and lactogenesis. J Clin Endocrinol Metab 1980;50:670–673. [PubMed: 7364925]
12. Cox DB, Owens RA, Hartmann PE. Blood and milk prolactin and the rate of milk synthesis in women. Exp Physiol 1996;81:1007–1020. [PubMed: 8960706]
13. Lauersen NH, Merkatz IR, Tejani N, Wilson KH, Roberson A, Mann LI, Fuchs F. Inhibition of premature labor: a multicenter comparison of ritodrine and ethanol. Am J Obstet Gynecol 1977;127:837–845. [PubMed: 851140]

14. Fuchs AR, Husslein P, Sumulong L, Micha JP, Dawood MY, Fuchs F. Plasma levels of oxytocin and 13,14-dihydro-15-keto prostaglandin F2 α in preterm labor and the effect of ethanol and ritrodine. Am J Obstet Gynecol 1982;144:753–759. [PubMed: 7148897]
15. Cobo E. Effect of different doses of ethanol on the milk-ejecting reflex in lactating women. Am J Obstet Gynecol 1973;115:817–821. [PubMed: 4688584]
16. Mennella JA. Short-term effects of maternal alcohol consumption on lactational performance. Alcohol Clin Exp Res 1998;22:1389–1392. [PubMed: 9802517]
17. 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]
18. Mennella JA, Beauchamp GK. Effects of beer on breast-fed infants. JAMA 1993;269:1637–1638. [PubMed: 8455295]
19. Bowen-Jones A, Thompson C, Drewett RF. Milk flow and sucking rates during breast-feeding. Dev Med Child Neurol 1982;24:626–633. [PubMed: 7141119]
20. Mennella JA. Infants′ suckling responses to the flavor of alcohol in mothers′ milk. Alcohol Clin Exp Res 1997;21:581–585. [PubMed: 9194908]
21. Díaz S, Serón-Ferré M, Cárdenas H, Schiappacasse V, Brandeis A, Croxatto HB. Circadian variation of basal prolactin, prolactin response to suckling, and length of amenorrhea in nursing women. J Clin Endocrinol Metab 1989;68:946–955. [PubMed: 2715293]
22. Little RE, Worthington-Roberts B, Mann SL, Uhl CN. Test-retest reliability of diet and drinking estimates for pregnancy and post partum. Am J Epidemiol 1984;120:794–797. [PubMed: 6496457]
23. Teff KL, Mattes R, Engelman K. Cephalic phase insulin release in normal weight males: verification and reliability. Am J Physiol 1991;261:E430–E436. [PubMed: 1928335]
24. Widstrom AM, Winberg J, Werner S, Hamberger B, Eneroth P, Uvnas-Moberg K. Suckling in lactating women stimulates the secretion of insulin and prolactin without concomitant effects on gastrin, growth hormone, calcitonin, vasopressin or catecholamines. Early Hum Dev 1984;10:115–122. [PubMed: 6389080]
25. Vuorenkoski V, Wasz-Hockert O, Koivisto E, Lind J. The effect of cry stimulus on the temperature of the lactating breast of primipara. A thermo-graphic study. Experientia 1969;25:1286–1287.
26. Grayson RH, Halperin JM, Sharma V, Schwartz ST, Koda VH, Newcorn JH. Changes in plasma prolactin and catecholamine metabolite levels following acute needle stick in children. Psychiatry Res 1997;69:27–32. [PubMed: 9080542]
27. Holdstock L, Wit H. Individual differences in the biphasic effects of ethanol. Alcohol Clin Exp Res 1988;22:1903–1911. [PubMed: 9884132]
28. O′Connor S, Morzorati S, Christian J, Li TK. Clamping breath alcohol concentration reduces experimental variance: application to the study of acute tolerance to alcohol and alcohol elimination rate. Alcohol Clin Exp Res 1998;22:202–210. [PubMed: 9514308]
29. Mumenthaler MS, Taylor JL, Yesavage JA. Ethanol pharmacokinetics in white women: nonlinear model fitting *versus* zero-order elimination analyses. Alcohol Clin Exp Res 2000;24:1353–1362. [PubMed: 11003200]
30. InnsPMorrisonPJPajoumondKEvaluation of fuel cell alcometer for forensic and pharmacokinetic purposes. Br J Clin Pharmacol19797439P440PProceedings
31. Mennella JA, Beauchamp GK. Beer, breast feeding and folklore. Dev Psychobiol 1993;26:459–466. [PubMed: 8293892]
32. Fischman MW, Schuster CR. Cocaine effects in sleep-deprived humans. Psychopharmacology (Berl) 1980;72:1–8. [PubMed: 6780998]
33. Gabriel K, Hoffman C, Glavas M, Weinberg J. The hormonal effects of alcohol use on the mother and fetus. Alcohol Health Res World 1998;22:170–177. [PubMed: 15706792]
34. Gianoulakis C. Alcohol-seeking behavior: the roles of the hypothalamic-pituitary adrenal axis and the endogenous opioid system. Alcohol Health Res World 1998;22:202–210. [PubMed: 15706797]
35. Chatterton RT Jr, Hill PD, Aldag JC, Hodges KR, Belknap SM, Zinaman MJ. Relation of plasma oxytocin and prolactin concentrations to milk production in mothers of preterm infants: influence of stress. J Clin Endocrinol Metab 2000;85:3661–3668. [PubMed: 11061519]

36. Gimpl G, Fahrenholz F. The oxytocin receptor system: structure, function and regulation. Physiol Rev 2001;81:629–683. [PubMed: 11274341]

37. Freeman ME, Kanyicska B, Lerant A, Nagy G. Prolactin: structure, function, and regulation of secretion. Physiol Rev 2000;80:1523–1631. [PubMed: 11015620]

38. De A, Boyadjieva N, Oomizu S, Sarkar DK. Ethanol induces hyper-prolactinemia by increasing prolactin release and lactotrope growth in female rats. Alcohol Clin Exp Res 2002;26:1420–1429. [PubMed: 12351938]

39. Pepino MY, Mennella JA. Advice given to women living in Argentina about breastfeeding, alcohol consumption and feeding practices. Rev Panam Salud Publica 2004;16:408–414. [PubMed: 15673483]

40. American Academy of Pediatrics Committee on Drugs. Transfer of drugs and other chemicals into human milk. Pediatrics 2001;108:776–789. [PubMed: 11533352]

41. Mennella, JA. Alcohol use during lactation: effects on the mother and breastfeeding infant. In: Watson, R., editor. Nutrition and alcohol. 2nd ed.. CRC Press; Boca Raton, FL: 2004. p. 377-391.

42. Zumoff B. Biological and endocrinological insights into the possible breast cancer risk from menopausal estrogen replacement therapy. Steroids 1993;58:196–204. [PubMed: 8395097]

43. Murrell TG. The potential for oxytocin (OT) to prevent breast cancer: a hypothesis. Breast Cancer Res Treat 1995;35:225–229. [PubMed: 7647345]

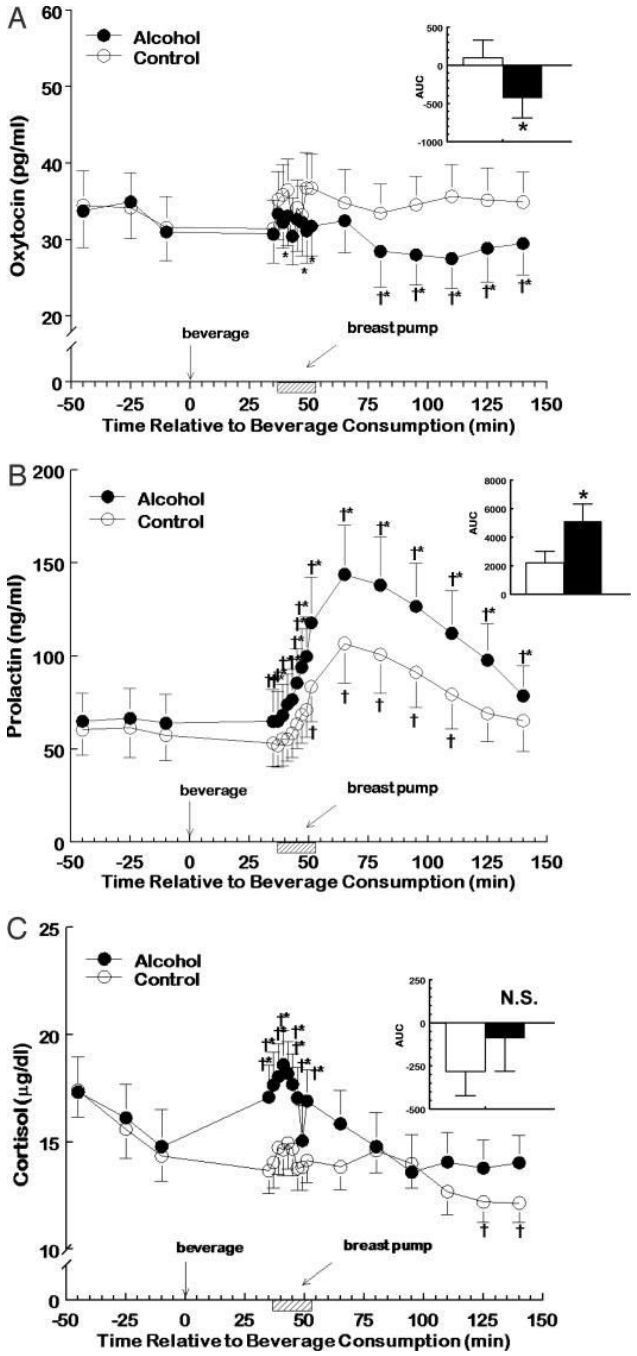

Fig. 1.

Mean (±SEM) plasma oxytocin levels (pg/ml; A), prolactin (ng/ml; B), and cortisol (μg/dl; C) in 17 lactating women at baseline and at varying times after the consumption of a 0.4-g/kg dose of alcohol in orange juice on one test day (•) and orange juice alone on the other (○). Women received breast stimulation with a breast pump (*hatched bars*) 35-51 min after the consumption of the beverage (time point = 0 min). The *inset* in each panel depicts mean (±SEM) AUC on alcohol (■) and control (□) days. *, Values that were significantly different from control session (AUC data) or significantly different from similar time points during the control (plasma levels; $P < 0.05$). †, Values within each test session that were significantly different from their respective baseline values ($P < 0.05$). Conversions are as follows: oxytocin in picograms per

milliliter × 0.80 = oxytocin in picomoles per liter; prolactin in nanograms per milliliter × 43.5 = prolactin in picomoles per liter; and cortisol in micrograms per deciliter × 27.6 = cortisol in nanomoles per liter.

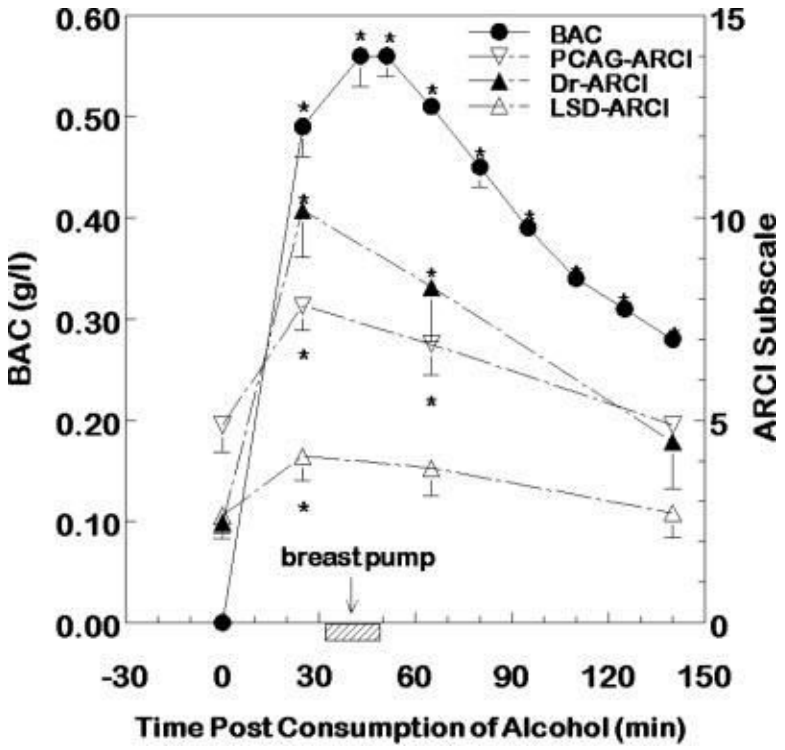

Fig. 2.

Mean (±SEM) estimated BAC (•) and raw scores of the ARCI to assess various measures of self-reported drug effects. The PCAG (▽), the LSD (△), and the Drunk Scale (▲) at baseline and at various time points after the consumption of a 0.4-g/kg dose of alcohol in 17 lactating women. The PCAG-ARCI scale provides a measure of sedation; the LSD-ARCI scale reflects dysphoria and somatic effects; and the Dr-ARCI scale reflects levels of drunkenness. To convert values for BAC to millimoles per liter, multiply by 0.217. *, Values that are significantly different from their respective baseline values ($P < 0.05$).

TABLE 1.

Schedule of events

					Begin BVG	End BVG		Breast pump stimulation				
Time (min)	**−85**	**−40**	**−25**	**−10**	**0**	**10**	**25**	**35–51**	**65**	**80**	**95**	**110**	**1**
Blood sampling		X	X	X				XXXXXXXXX	X	X	X	X	
BAC	X						X	X X	X	X	X	X	
ARCI mood ratings	X						X		X				

Each subject was tested on 2 d separated by 1 wk. On both test days, subjects consumed a beverage (BVG) at time 0. On one test day, the BVG was a 0.4-g/kg dose of alcohol in orange juice (alcoholic BVG), whereas on the other test day the BVG was an equal volume of orange juice (control BVG). Approximately 35 min after drinking the BVG, the women pumped their breasts with an electric breast pump for 16 min (breast stimulation). The symbol X denotes occurrence of blood sampling, determination of BAC, and completion of ARCI questionnaires to evaluate various mood states before and after the consumption of the BVG.

TABLE 2.

Correlations among and between oxytocin and prolactin AUCs during breast stimulation and lactational performance on the control day when lactating women consumed a nonalcoholic beverage and the alcohol day when they comsumed a 0.4-g/kg dose of alcohol

	Control day			Alcohol day			
	AUC Prolactin	**Milk ejection latency (sec)**	**Milk production (ml)**	**AUC oxytocin**	**AUC prolactin**	**Milk ejection latency (sec)**	**Milk production (**
Control day							
AUC oxytocin	0.01 $P = 0.96$	−0.12 $P = 0.63$	0.12 $P = 0.65$	0.80 $P < 0.01$	−0.02 $P = 0.93$	0.23 $P = 0.38$	−0.07 $P = 0.78$
AUC prolactin		0.17 $P = 0.52$	−0.11 $P = 0.67$	−0.19 $P = 0.46$	0.28 $P = 0.28$	−0.14 $P = 0.58$	−0.11 $P = 0.69$
Milk ejection latency (sec)			0.08 $P = 0.75$	−0.11 $P = 0.67$	0.11 $P = 0.66$	0.42 $P = 0.91$	0.13 $P = 0.61$
Milk production (ml)				0.21 $P = 0.41$	−0.12 $P = 0.65$	−0.14 $P = 0.59$	0.72 $P < 0.01$
Alcohol day							
AUC oxytocin					−0.06 $P = 0.81$	−0.05 $P = 0.86$	0.23 $P = 0.38$
AUC prolactin						0.49 $P = 0.045$	0.22 $P = 0.40$
Milk ejection latency (sec)							−0.32 $P = 0.21$