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Julie A. Mennella and Marta Yanina Pepino

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# Breastfeeding and Prolactin Levels in Lactating Women With a Family History of Alcoholism

**WHAT'S KNOWN ON THIS SUBJECT:** Studies indicate that men and nonlactating women with a family history of alcoholism but no alcoholism themselves display blunted prolactin responses to an alcohol challenge when compared with individuals without a family history; however, there are no such studies of lactating women.

**WHAT THIS STUDY ADDS:** Family history of alcoholism is associated with blunted magnitude, rapidity, and duration of prolactin responses to breast stimulation and an alcohol challenge in lactating women. More frequent breastfeeding by FHP women suggests behavioral compensation for perceived and/or actual poor lactation.


## abstract

**OBJECTIVE:** Many motivated new mothers fail to reach public health goals for breastfeeding, highlighting the need to identify risk factors. Because having a family history of alcoholism is associated with blunted prolactin responses to an alcohol challenge in nonlactating individuals, this study aimed to identify associations in family history of alcoholism, prolactin, and breastfeeding behaviors in lactating women.

**METHODS:** This was a 2-day experimental study that used within-subject alcohol or control beverage consumption and between-subject family history of alcoholism factors. The participants were non–alcohol-dependent lactating women; 7 were family history–positive (FHP) for alcohol dependence, and 21 were family history–negative (FHN). Consumption of 0.4 g/kg alcohol or nonalcoholic beverage occurred in separate randomized sessions, followed by use of a breast pump. Basal and suckling-induced prolactin, blood alcohol concentrations, milk yield, self-reported drug effects, neophobia, and breastfeeding patterning were measured.

**RESULTS:** Although no group differences in alcohol pharmacokinetics were detected, FHP women exhibited blunted prolactin to breast stimulation after drinking the control and alcohol beverage and felt more of the stimulant-like effects of alcohol than did FHN women. FHP women reported more frequent daily breastfeeding than did FHN women.

**CONCLUSIONS:** This is the first evidence that family history of alcoholism is associated with a blunted magnitude, rapidity, and duration of the prolactin response to breast stimulation and an alcohol challenge in lactating women. More frequent breastfeeding by FHP women suggests behavioral compensation for perceived and/or actual poor lactation. Alcohol did not enhance lactational performance, further disputing the lore that alcohol is a galactagogue. *Pediatrics* 2010;125:e1162–e1170



**AUTHORS:** Julie A. Mennella, PhD and Marta Yanina Pepino, PhD

*Monell Chemical Senses Center, Philadelphia, Pennsylvania*



**KEY WORDS**
lactation, breastfeeding, alcohol, risk factors, endocrinology, prolactin, drug effects


**ABBREVIATIONS**
FHP—family history–positive
FHN—family history–negative
BAC—blood alcohol concentration
AUC—area under the curve


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Address correspondence to Julie A. Mennella, PhD, Monell Chemical Senses Center, 3500 Market St, Philadelphia, PA 19104-3308. E-mail: mennella@monell.org



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Scientific evidence overwhelmingly indicates that breastfeeding confers significant health benefits to mother and child and is the ideal method for feeding and nurturing infants. The US public health goals are for at least 75% of the nation's new mothers to breastfeed at hospital discharge and at least 50% to breastfeed throughout the first 6 months of their child's life.[1] Although a variety of social and cultural factors may influence a woman's decision to breastfeed, even many highly motivated women fail to reach these goals.[2] Consequently, there has been great interest in identifying risk factors for poor breastfeeding performance and mechanisms that can be targeted by supportive interventions.

Prolactin is one of the principal lactogenic hormones, and blunted prolactin response to suckling has previously been identified as a major mechanism of breastfeeding failure during the early stages of lactation.[3] Suckling, the most potent physiologic stimulus of prolactin, upregulates opioids and other factors that inhibit dopamine secretion into the portal circulation.[4,5] Dopamine inhibitors, such as phenothiazines, metoclopramide, domperidone, and alcohol, also cause hyperprolactinemia,[6] and some are used specifically as galactagogues.[7]

Encouraging lactating women to drink alcohol as a means to increase milk production is a widespread folklore[8,9] that is still perpetuated by early evidence, albeit in men and nonlactating women, that alcohol consumption can increase circulating prolactin[10,11]; however, the first evidence that alcohol influences lactation were reports that maternal alcohol intake decreased the milk intake of rat pups.[12,13] Consistent with these animal studies, we have shown that after lactating women consume a moderate dose of alcohol, their infants breastfeed less despite sucking more intensely during the initial minutes of feeding.[14,15] This is, at least in part, because moderate doses of alcohol, despite stimulating prolactin release, interfere with milk ejection in a time-dependent manner and milk production during a 4- to 5-hour period.[16–18]

A number of studies indicated that men and nonlactating women who had a history of alcohol dependence in first- or second-generation family members but were not themselves alcoholics were less acutely influenced by the sedative and motor effects of alcohol than are individuals without such a family history.[19–21] Although alcohol consumption results in increased prolactin levels, individuals with a familial history of alcoholism exhibit a blunted prolactin response to alcohol,[19,20] suggesting that family history alone may be an important indicator of prolactin dynamics; however, this research was conducted in men and nonlactating women, and, to the best of our knowledge, there have been no such studies of lactating women.

This study tests the hypothesis that lactating women who have a family history of alcoholism but themselves are not alcoholics exhibit smaller prolactin responses to suckling and an alcohol challenge than do women without such a family history. We focused on women in established lactation. Although relative differences in prolactin responses to suckling in established lactation are likely to reflect hormonal responses in early lactation,[22] prolactin is not critical in established lactation as it is in early lactation. We therefore hypothesized that if family history of alcoholism has a negative impact on prolactin responses, then these women are likely to exhibit compensatory changes in breastfeeding patterning that will be evident during established lactation, and despite alcohol-induced elevations in prolactin, alcohol consumption will not enhance lactational performance.

## METHODS

### Study Population

The study population consisted of 28 healthy lactating women who participated in 1 of 2 research studies at the Clinical and Translational Research Center at the University of Pennsylvania.[17,18] Because similar interventions and data collection methods were used in the 2 studies, the populations were combined for these analyses. Eligible participants were women of any race who were exclusively nursing (no solid food or formula feeding) 2- to 5-month-old infants and who had experience using a breast pump. Exclusion criteria were pregnancy, obesity, smoking, anemia, alcohol dependence or lifetime alcohol abstinence, gastric bypass surgery, resumption of menses, or use of any medication including oral contraceptives.

Participants were classified as having a family history of alcoholism (family history–positive [FHP]) or having no family history of alcoholism (family history–negative [FHN]) by using the Family Interview for Genetic Studies according to the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition* criteria for family members up to first-degree relatives.[23] The groups were matched for parity to account for reductions in circulating prolactin associated with previous child birth.[24] Women abstained from alcohol for at least 3 days before testing and fasted the night before and during each testing session, because prolactin concentrations can be elevated by certain gastrointestinal hormones and high blood glucose.[25] All testing procedures were approved by the Office of Regulatory Affairs at the University of Pennsylvania, and each woman gave informed written consent before testing.


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## Interventional Procedures

Participants attended 2 test sessions separated by 1 week, in which measurements were made after consumption of either an alcoholic or a nonalcoholic beverage. Each test session was conducted at the same time of day to account for the circadian rhythm of prolactin release.[26] Participants attended the study without their infants and were not allowed to engage in activities that might influence prolactin levels, including sleeping, talking about infants or food, or watching television (which could expose them to images of infants and food). Participants were weighed and were confirmed to be not pregnant and to have fasted, after which an indwelling catheter was inserted into the antecubital vein. Because prolactin is stress labile,[27] participants acclimated in the private testing room for at least 45 minutes before any interventions.

In randomized order, women drank a 0.4-g/kg dose of alcohol in orange juice (15% v/v) during 1 test session (alcohol condition) and an equal volume of orange juice (control condition) during the other, having last breastfed their infants at home 3.5 ± 0.3 hours earlier. Thirty-five minutes after consuming the beverage (when blood alcohol concentration [BAC] is expected to peak), participants used an electric breast pump (Medela Symphony, McHenry, IL) for 16 minutes, alternating the breast being pumped every 2 minutes. The total amount of milk pumped during this period was measured. The use of a breast pump avoided variation in suckling intensity applied by infants[14,28] and changes in prolactin and milk let-down associated with olfactory, visual, or auditory cues elicited by infants.[29]

BAC was determined by having the participant breathe into an Alco-Sensor IV (Intoximeter, Inc, St Louis, MO), and blood samples were collected before (−10 minutes) and at fixed intervals after (35, 37, 39, 41, 43, 45, 47, 49, 51, 65, 80, 95, 110, 125, and 140 minutes) beverage consumption. Blood samples were collected, after discarding the first 1 mL, into EDTA tubes. After the last collection, participants were fed and allowed to leave the center when BAC levels registered 0.

## Alcohol Pharmacokinetics and Subjective Responses

Peak BAC, time to peak BAC, and alcohol disappearance rates ($\beta_{60}$; g/L per h) were calculated. $\beta_{60}$ was determined from the slope of the linear least-squares regression of the apparent descending limb of the BAC time curve.[30] Participants completed the Addiction Research Center Inventory at fixed intervals before and after drinking the beverages.[31] This questionnaire comprises a number of scales that measure drug-induced euphoria, sedation, dysphoric and somatic effects, stimulant-like effects, and drunkenness.

## Analyses of Blood Prolactin

Prolactin levels in plasma samples were measured in duplicate by a direct, 2-site immunoradiometric assay without extraction, using materials supplied by ICN Diagnostics (Costa Mesa, CA). The antiserum used in this assay cross-reacts <0.01% with human chorionic gonadotropin, thyrotropin, luteinizing hormone, and follicle-stimulating hormone. The minimal detectable concentration was 0.19 ng/mL, and the intra-assay and interassay variations were 5.4% and 10.1%, respectively, across all assays performed.

The area under the plasma prolactin time curve (AUC; μg/min per L) was determined by using the trapezoid rule (OriginLab Corp, Northampton, MA) for 2 time periods: during ($AUC_{35–51}$) and after ($AUC_{51–140}$) breast pumping stimulation. Prolactin levels just before pumping were used as baseline values. Linear least-squares regression of the apparent ascending limb of the curve of prolactin response to breast stimulation was used to determine the rate of the prolactin rise (slope) for each beverage condition.

## Personality and Behavioral Assessments

Several days before each testing day, all but 1 participant kept a log describing how often and at what time of day they nursed their infants. Because some personality traits (eg, depression, social phobia)[32] as well as subtle psychological/personality changes that occur during the postpartum period[33] correlate with prolactin, each participant completed questionnaires to determine their propensity to try or avoid novel foods (food neophobia) and unwillingness to experience new situations and people, typically expressed as a "desire to get home to my familiar surroundings" or "to feel uneasy in unfamiliar surrounding" (general neophobia).[34]

## Statistical Analysis

The primary variables were prolactin response to breast pumping and alcohol, milk yield during a standard 16-minute pumping period, and self-reported patterning (frequency and timing) of nursing. Secondary variables included neophobia and subjective effects of alcohol. Two main analyses were conducted to determine the effects on these variables of having a family history of alcoholism. First, the influence of family history of alcoholism in the absence of alcohol consumption was determined. Second, whether moderate drinking differentially altered these variables as a function of family history of alcoholism was determined.



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For each measure, repeated-measures analyses of variance were conducted with family history of alcoholism (FHP, FHN) as a between-subject factor and beverage condition (alcohol, control) and time since beverage consumption (when applicable) modeled as within-subject repeated-measure factors. When significant variation was detected, posthoc Fisher least significant difference analyses were conducted. Pearson correlation coefficients were conducted to determine whether relationships existed between prolactin, BAC, neophobia, and subjective effects. Separate analyses of variance or $\chi^2$ analyses were conducted to determine whether there were group differences in demographics and patterning of breastfeeding. All analyses were performed with Statistica 8.0 (StatSoft, Tulsa, OK), and the criterion for statistical significance was $P < .05$.

## RESULTS

### Participant Characteristics

Recruited participants were non–alcohol-dependent lactating women (29% black, 54% white, and 17% other) whose ages ranged from 21 to 40 years (mean: 32.7 ± 1.0 years). Women who had a first-degree relative with a history of alcohol dependence were categorized as FHP ($n = 7$), whereas those who had no history of alcoholism in the first generation were categorized as FHN ($n = 21$). As shown in Table 1, the 2 groups were of similar age, number of months postpartum (consequently, their infants were of similar age), parity, BMI, and drinking habits. As a group, women reported that alcohol intake was low during pregnancy but significantly increased during lactation (0.3 ± 0.9 vs 1.5 ± 0.3 standard drinks per month; $P = .001$); however, there were no significant differences between FHN and FHP women in the amount of alcohol consumed during pregnancy or lactation. Neither were there group differences in the increment of alcohol intake from the prepartum to postpartum periods.

**TABLE 1** Participant Characteristics

| Characteristic | Participant Group | | *P* |
|---|---|---|---|
| | FHN | FHP | |
| Age, mean ± SD, y | 31.5 ± 1.1 | 35.2 ± 1.9 | .10 |
| BMI, mean ± SD, kg/m$^2$ | 25.2 ± 0.9 | 25.1 ± 1.6 | .97 |
| Parity, % nulliparous | 43 | 43 | .67 |
| Time postpartum, mean ± SD, mo | 3.1 ± 0.2 | 3.5 ± 0.3 | .28 |
| Age started drinking, mean ± SD, y | 17.0 ± 0.7 | 17.0 ± 1.2 | .97 |
| Alcohol consumption during past 3 wk | | | |
| No. of standard drinks | 1.2 ± 0.4 | 2.2 ± 0.7 | .26 |
| No. of drinking occasions | 0.9 ± 0.3 | 1.3 ± 0.5 | .54 |
| Food neophobia score | 32.4 ± 2.4 | 28.4 ± 4.1 | .41 |
| General neophobia score | 27.6 ± 1.5 | 20.6 ± 2.6 | .03[a] |
| No. of times infants breastfed | | | |
| 8:00 AM and 1:00 PM | 2.5 ± 0.2 | 2.8 ± 0.4 | .51 |
| 1:00 PM to 8:00 AM | 6.2 ± 0.4 | 8.1 ± 0.8 | .04[a] |
| No. of participants | 21 | 7 | |

[a] Significance of difference between FHP and FHN lactating women.

### Prolactin Responses

There were no differences in baseline ($t = 35$ minutes) prolactin concentrations between the 2 groups (FHP and FHN). The suckling stimulus that was used in this study, 16 minutes of breast pumping, effectively increased plasma prolactin concentrations ($P < .00001$), although the magnitude, rapidity, and duration of the prolactin responses differed between the 2 groups ($P = .03$). As shown in Fig 1A, breast stimulation in FHN women was associated with significantly elevated prolactin concentrations compared with baseline values, beginning just 10 minutes after the start of breast pumping ($t = 45$ minutes) and lasting for 90 minutes ($t = 135$ minutes). Although a similar prolactin response to breast pumping was observed in FHP participants, the values rose significantly above baseline only 30 minutes after the start of breast pumping ($t = 65$ minutes) and had returned to baseline shortly thereafter ($t = 80$ minutes).

Figure 1B shows that in both groups of women, alcohol magnified the prolactin response to breast pumping. Prolactin increased sooner and remained elevated longer after the initiation of breast pumping when women had drunk the alcohol compared with the control beverage; however, the effect of alcohol on the prolactin response to pumping differed between the FHN and FHP participants ($P < .0001$).

For both groups, the magnitude of the prolactin response to breast stimulation was significantly larger on the alcohol than the control day ($AUC_{35–51}$: $P = .02$; $AUC_{51–140}$: $P = .04$; Table 2); however, regardless of the beverage condition, there was a main effect of family history on both AUC and peak prolactin values. FHP women exhibited a smaller prolactin response to breast stimulation compared with FHN women (prolactin peak, $AUC_{35–51}$, $AUC_{51–140}$: all $P < .05$). Furthermore, although the linear prolactin rise was steeper when FHN women drank the alcoholic beverage than the control beverage ($P = .04$), there was no effect of beverage condition on the slope of the prolactin rise in FHP women. Despite these effects of alcohol consumption on prolactin and differences between FHP and FHN participants, no significant correlations were identified between prolactin and subjective effects of alcohol.


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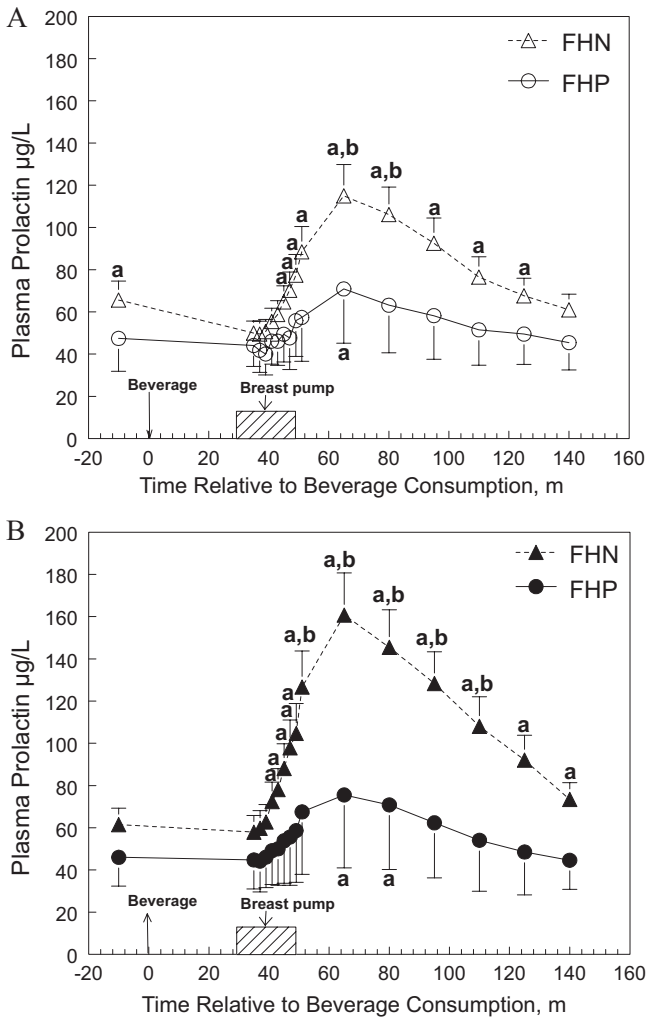


**FIGURE 1**

Mean ± SEM plasma prolactin ($\mu$g/L) among FHP (circles) and FHN (triangles) lactating women at baseline and at varying times after consumption of orange juice alone in 1 test session (open symbols; A) and a 0.4-g/kg dose of alcohol in orange juice in the other (closed symbols; B). Women received breast stimulation with a breast pump (▨) 35 to 51 minutes after the consumption of the beverage (time point = 0). [a] Values within each test session that are significantly different from their respective baseline values ($P < .05$). [b] Values that are significantly different from similar time points between FHP and FHN women (plasma levels; $P < .05$). To convert prolactin in micrograms per liter to picomoles per liter, multiply by 43.5.

There was no significant effect of family history (FHN versus FHP: 110.7 ± 11.3 vs 86.6 ± 19.6 mL; $P = .30$) or beverage condition (control versus alcohol: 100.8 ± 11.2 vs 96.5 ± 12.7 mL; $P = .58$) or interaction between these variables on how much milk the women pumped during the 16-minute pumping session. Neither were there significant relationships between milk yield and the prolactin AUCs, prolactin slope, or peak prolactin concentrations on either the control or the alcohol day.

### Maternal Behavioral Characteristics and Breastfeeding Patterning

Because some personality traits (eg, social phobia)[32] as well as subtle psychological changes that occur during the postpartum period[33] have been shown to correlate with prolactin responsivity, we investigated whether there were differences in neophobia. Although the 2 groups were similar in their willingness to try new foods, Table 1 shows that FHP lactating women were less neophobic than FHN lactating women ($P = .03$). Neophobia was positively correlated with the magnitude of the peak prolactin response to breast pumping ($P < .001$; Fig 2).

FHP women reported nursing their infants more frequently ($P < .05$) than those who were FHN. Although both groups of women nursed their infants on average every 2 hours between 8:00 AM and 1:00 PM (the time at which the study occurred; $P = .51$), FHP women nursed more often during late afternoon to early morning hours than did FHN women ($P < .05$; Table 1).

### Subjective Effects and Pharmacokinetics of Alcohol

As shown in Fig 3, there were no significant differences in the rising and falling BAC curves between FHP and FHN women. For both groups, BAC peaked at ~45 ± 2 minutes after the consumption of the alcoholic beverage and decreased thereafter ($P < .0001$). The total amount of alcohol eliminated ($b_{60}$) was 5.9 ± 0.4 g/h; the elimination rate was 0.09 ± 0.01 g/kg per h, and the disappearance rate ($\beta_{60}$) was 0.15 ± 0.01 g/L per h. In both groups, alcohol consumption increased feelings of sedation ($P < .0001$), dysphoria ($P < .001$), and drunkenness ($P < .0001$) over time; however, FHP women felt greater stimulant effects of alcohol ($P = .02$) and more euphoria ($P = .06$) than did FHN women.

## DISCUSSION

This is the first experimental evidence that in lactating women, a family history of alcoholism in first-degree relatives is a determinant of hormonal and behavioral responses to breast stimulation and alcohol consumption.



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**TABLE 2** Effects of Family History of Alcoholism and Beverage Condition on Prolactin Responses to Breast Pumping

| Prolactin Responses | FHN | | | FHP | | |
|---|---|---|---|---|---|---|
| | Control | Alcohol | Both Beverages Combined | Control | Alcohol | Both Beverages Combined |
| Baseline prolactin | 50.0 ± 5.7 | 57.8 ± 7.9 | 53.9 ± 6.1 | 44.1 ± 9.9 | 44.7 ± 13.8 | 44.4 ± 10.6 |
| Slope of prolactin rise[a] | 2.6 ± 0.5 | 4.6 ± 0.7[b,c] | 3.6 ± 0.6 | 1.5 ± 1.2 | 1.2 ± 0.9 | 1.3 ± 1.0[d] |
| Prolactin AUC, $\mu$ g/min per L | | | | | | |
| 35–51 min[a] | 215.4 ± 49.3 | 385.9 ± 63.9[b,c] | 300.5 ± 51.8 | 50.2 ± 85.4 | 111.41 ± 110.6[b,c] | 80.8 ± 89.8[b,e] |
| 51–140 min[a] | 3415.2 ± 664.1 | 5729.9 ± 844.0 | 4572.1 ± 695.3 | 1181.4 ± 1150.2 | 1457.5 ± 1461.8 | 1319.5 ± 1204.4[b,e] |
| Peak prolactin, $\mu$ g/L[f] | 118.0 ± 14.5 | 163.2 ± 20.0 | 140.6 ± 16.2 | 70.1 ± 25.1 | 75.9 ± 34.7 | 73.0 ± 28.1[b,e] |

Data are means ± SEM.

[a] $P < .05$ for the comparison between alcohol and control (family history combined); data not shown.

[b] Significant effects are indicated by bold fonts.

[c] $P < .05$ for the comparison within Beverage Conditions, Same Family History.

[d] $P = .06$ for the comparison with FHN (beverage conditions combined).

[e] $P < .05$ for the comparison with FHN (beverage conditions combined).

[f] $P = .06$ for the comparison between alcohol and control (family history combined); data not shown.

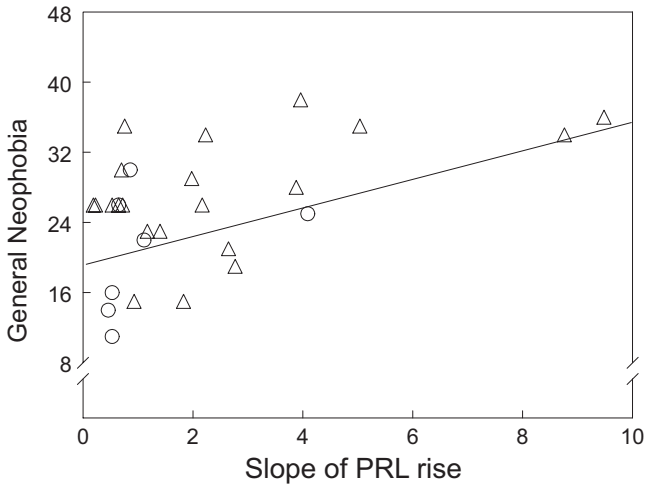


**FIGURE 2**
Relationship between the slopes of the prolactin rise to breast pumping during the control condition and neophobia scores among FHP (○) and FHN (△) lactating women ($r = 0.51$, degrees of freedom = 27, $P < .001$).

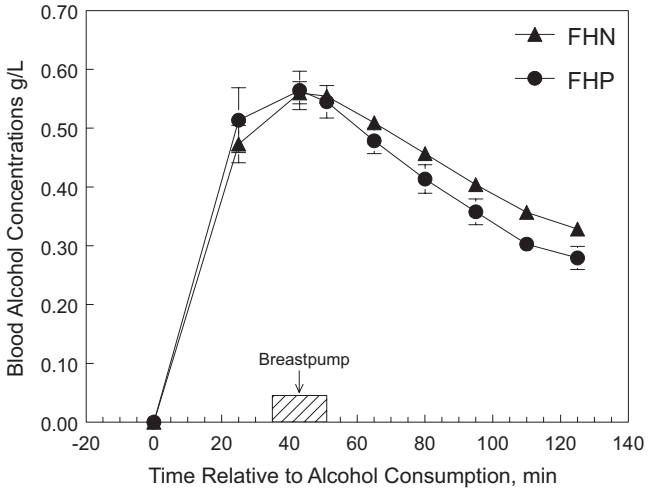


**FIGURE 3**
BAC curve for FHP (●) and FHN (▲) lactating women after drinking a 0.4-g/kg dose of alcohol. To convert values for BAC to millimoles per liter, multiply by 0.217.

FHN and FHP women did not differ in their baseline prolactin values, and both exhibited an increase in prolactin in response to breast stimulation, although later and for a shorter period of time in FHP women compared with FHN women. Whereas moderate alcohol consumption increased the rapidity and duration of the prolactin response to breast stimulation in all women, only FHN women exhibited an increase in peak prolactin response to breast stimulation after alcohol consumption. These differences were not attributable to differences in alcohol pharmacokinetics.

As expected, differences in the effects of alcohol consumption on prolactin responses between FHP and FHN lactating women were qualitatively consistent with but more robust than those reported between FHP and FHN men and nonlactating women.[19,20] FHP lactating women also exhibited increased stimulant-like effects of alcohol than did FHN women, despite similar BACs and drinking histories, as reported previously in FHP and FHN men and nonlactating women.[35] A higher subjective response to alcohol is associated with higher dopamine (D2) receptor availability in the nucleus accumbens.[36]

Although never studied in lactating women, it has been suggested that do-



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paminergic dysregulation, characterized by lower D2 receptor availability in the striatum[37] and larger prolactin responses, when challenged with dopamine antagonists[32] is associated with certain behavioral phobias. Maternal neophobia, which is known to increase during lactation, promotes focus on and protection of the infant.[33,38] Our study, which demonstrates a positive correlation between neophobia and prolactin response to breast stimulation, supports a neuroendocrine basis for maternal neophobia. Perhaps higher-than-normal levels of dopamine receptors in the caudate and striatum (in contrast to individuals with social phobia)[32] might explain the reduced neophobia and smaller prolactin responses when challenged with alcohol in FHP lactating women (whose levels of neophobia were similar to those of nulliparous women[31,39]). Taken together, these observations support the hypothesis that unaffected adult progeny from alcoholic pedigrees exhibit a fundamental dopaminergic dysregulation that affects personality-related differences in susceptibility of different brain areas.[40]

Familial history of alcoholism in nonalcoholic lactating women disrupts the lactational hormone milieu, resulting directly or indirectly in altered breastfeeding patterns during established lactation. This phenotype, a blunted prolactin response, is now considered an important risk factor for failure to initiate and sustain lactation among obese women,[3,41] perhaps as a result of the obesity-related alterations in dopaminergic neurotransmission.[42] Despite more persistent attempts to breastfeed in the first week postpartum,[3] obese women exhibit delayed secretory activation and, ultimately, premature cessation of breastfeeding than normal-weight women. As a consequence of this discovery, prolactin-promoting interventions, such as putting infants to the breast soon after birth and promoting frequent suckling, have substantially improved breastfeeding outcomes in obese women.[3]

Whether such prolactin-promoting strategies contributed to the breastfeeding success of the FHP women in this study, all of whom were of normal weight, is an important area for future research. Their more frequent breastfeeding during the afternoon and evening hours, the time of day when prolactin levels are naturally lowest because of circadian periodicity,[4,43] may suggest another prolactin-promoting intervention for women with blunted prolactin responses (perhaps more infant-led feeding patterning). In other words, a blunted prolactin response to suckling does not mean that a woman *cannot* successfully breastfeed. Regardless of the prolactin response, alcohol consumption did not result in greater milk production during the 16-minute pumping period, a finding that adds to the growing body of scientific literature that refutes the folklore that alcohol is a galactagogue.[14,15,17]

National estimates of the prevalence of family history of alcoholism in the United States are at least as high as those for adult obesity.[44,45] Whether the hormonal and behavioral differences noted in FHP women contribute to early weaning and whether FHP women warrant similar support during the early stages of lactation as do obese women are still unknown. Addressing the limitations that such familial factors may impose on breastfeeding performance and the development of evidence-based strategies that lead to lactation success will be useful to help new mothers overcome breastfeeding barriers and to health officials who attempt to devise targeted breastfeeding interventions as well as to provide sound guidelines for ethanol consumption during lactation.[46]

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Julie A. Mennella and Marta Yanina Pepino

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