


[Advertising Disclaimer »](#)

Pediatrics

May 2004, VOLUME 113 / ISSUE 5

Prepregnant Overweight and Obesity Diminish the Prolactin Response to Suckling in the First Week Postpartum

Kathleen M. Rasmussen, Chris L. Kjolhede

Article

Figures & Data

Info & Metrics


Comments

[Download PDF](#)

Abstract

Objective. The population subgroups with the highest proportion of overweight and obese women often are characterized by the lowest rates of initiation and shortest durations of breastfeeding. We previously documented that these 2 population-level trends may be related. In a population of white women who lived in a rural area, we observed that prepregnant overweight and obesity were associated with failure to initiate and also to sustain lactation. The means by which being overweight or obese negatively affect lactational performance is unknown and likely to be multifactorial in origin, including the simple mechanical difficulties of latching on and proper positioning of the infant. In addition, we have shown that prepregnant body mass index (BMI) is negatively associated with the timing of lactogenesis II, the onset of copious milk secretion. Although the effects of obesity on the prolactin response to infant suckling have never been studied, we postulated that maternal obesity could compromise this important response. We proposed that this might occur because obesity alters the 24-hour spontaneous release of prolactin and also because prolactin secretion is blunted in response to various stimuli among obese subjects. The fall in progesterone

 **Pediatric Care Online™**
Treat with confidence. Trusted answers from the American Academy of Pediatrics



Trusted advice.
Always available.
Immediate access to
NEW App
NEW eTextbook
NEW eReference
of Pediatric Care

supported by
MeadJohnson
Nutrition

American Academy
of Pediatrics
DEDICATED TO THE HEALTH OF ALL CHILDREN

[Advertising Disclaimer](#)



View this article with
LENS



Previous

Next 



Email



Share

concentration that occurs immediately postpartum is the trigger for the onset of copious milk secretion, but maintenance of prolactin and cortisol concentrations is necessary for this trigger to be effective. Adipose tissue concentrates progesterone. We proposed that this additional source of progesterone would lead to consistently higher progesterone concentrations among obese compared with normal-weight women. This, in turn, would lead to a delay in reaching the appropriate concentration to trigger the onset of lactogenesis II. We tested the hypotheses that a reduced prolactin response to suckling and higher-than-normal progesterone concentration in the first week after delivery might be among the means by which maternal overweight could compromise early lactation.

Methods. We enrolled 40 mothers of term infants from the same population that we studied previously. We measured serum prolactin and progesterone concentrations by radioimmunoassay before and 30 minutes after the beginning of a suckling episode at 48 hours and 7 days after delivery. We used path analysis to develop a parsimonious multivariate prediction of the prolactin response to suckling at 48 hours and 7 days postpartum.

Results. As expected, prolactin values decreased from 48 hours to 7 days postpartum. Women who were overweight or obese (using the Institute of Medicine's cutoff for women of a BMI >26 kg/m²) before conception had a lower prolactin response to suckling than normal-weight women at 48 hours but not at day 7. In multivariate analyses, overweight/obesity, primiparity, and birth weight were negatively associated with the prolactin response to suckling at 48 hours. After adjustment for confounding by time since delivery and the duration of the nursing episode, only overweight/obesity remained a significant negative predictor of prolactin response to suckling at day 7. Concentrations of progesterone decreased dramatically from 48 hours to 7 days postpartum but did not differ between normal-weight and overweight/obese women at either time. In addition, the decreases in progesterone concentrations from 48 hours to 7 days postpartum did not differ between the prepregnant BMI groups.

Conclusion. The unique and important finding from this study is that overweight/obese women had a lower prolactin response to suckling. This would be expected to compromise the ability of overweight/obese women to produce milk and, over time, could lead to premature cessation of lactation. These findings are important because, during our observation

 Permissions

 Print

 Alerts

 PDF

 Citation Tools

Table of Contents

[Early Release](#)

[Current Issue](#)

[Past Issues](#)

[AAP Policy & Collections](#)

[Editorial Board](#)

[Editorial Policies](#)

[Overview](#)

[Open Access](#)

[Author Guidelines](#)

[Reviewer Guidelines](#)

[Submit My Manuscript](#)

Subjects

[Endocrinology](#)

[Endocrinology](#)

[Fetus/Newborn Infant](#)

[Fetus/Newborn Infant](#)

[Obesity](#)

[Obesity](#)

Related Articles

Breastfeeding: the essential principles.
Latha Chandran et al., *Pediatr Rev*, 2006

Breastfeeding update. 2: Clinical lactation management.
N G Powers et al., *Pediatr Rev*, 1997

Delayed Lactogenesis II Decreases Breastfeeding Success
American Academy of Pediatrics, AAP Grand Rounds, 2012

Risk Factors for Suboptimal Infant Breastfeeding Behavior, Delayed Onset of Lactation, and

period (just before and after lactogenesis II, the time of onset of copious milk secretion), the prolactin response to suckling is more important for milk production than it is later in lactation. We have previously shown that a high proportion of the overweight and obese women in this population who give up on breastfeeding do so at this time. This finding thus provides evidence of a biological basis for this association, and additional study of it is likely to be informative. We postulated that there would be consistently higher progesterone concentrations in the early postpartum period among obese compared with normal-weight women because adipose tissue is an extraplacental source of this hormone. This hypothesis was not supported in this study because there were no significant differences between normal-weight and overweight/obese women in progesterone concentrations at either 48 hours or 7 days postpartum. The values that we observed at these times were similar to those reported by others in the early postpartum period. The findings from this study add plausibility to our observation that initiation, not just duration of breastfeeding, is negatively affected by maternal overweight/obesity. Although women should begin pregnancy at a healthy weight and gain reasonably during gestation, not all will. Pediatricians can help overweight/obese women to succeed at breastfeeding by targeting them for contact with a lactation consultant before discharge from the hospital to be sure that they have received optimal advice on breastfeeding techniques. In addition, early contact with the mother after discharge—by calling her at home to offer her support and counseling for breastfeeding, by scheduling the first pediatric visit earlier than for other patients, or by enlisting the assistance of public health nurses for a home visit if this is possible—would help overweight/obese women to continue to breastfeed. Being overweight or obese is negatively associated with the prolactin response to suckling in the first week postpartum and, thus, may contribute to early lactation failure.

[obesity](#) [prolactin](#) [breastfeeding](#) [lactogenesis](#) [estrogen](#)

[progesterone](#) [leptin](#)

Although we have made progress in meeting national goals for initiation of breastfeeding in recent years, we have made disturbingly little progress toward national goals for maintenance of breastfeeding in this same period¹—despite overwhelming scientific evidence that documents the benefits of this method of feeding infants.² Simultaneously, American women in general and some racial/ethnic subgroups in particular have

[Excess Neonatal Weight Loss](#)

Kathryn G. Dewey et al., *Pediatrics*, 2003

[Effects of recombinant human prolactin on breast milk composition.](#)

Camille E Powe et al., *Pediatrics*, 2011

[Recognizing and Treating Delayed or Failed Lactogenesis II](#)

Nancy M. Hurst et al., *Medscape*, 2007

[Heavier moms more likely to stop breastfeeding for certain reasons](#)

Univadis (UK), 2015

[Obesity, Insulin Treatment Contribute to Delayed Lactogenesis](#)

Medscape, 2013

[Breastfeeding and Adoption](#)

Dena Goffman, MD and Peter Bernstein, MD, MD, *Medscape*, 2005

[Diabetes Affects Breastfeeding; Will Metformin Help?](#)

Yael Waknine et al., *Medscape*, 2014

Powered by
Trend MD

gotten fatter.³ The population subgroups with the highest proportion of overweight and obese women often are characterized by the lowest rates of initiation and shortest durations of breastfeeding. We have previously documented that these 2 population-level trends may be related. In a population of white women who lived in a rural area, we observed that those who were overweight or obese before conception had a significantly increased risk of failing to initiate breastfeeding successfully.⁴ Those who did initiate breastfeeding successfully also stopped this practice significantly sooner than their normal-weight counterparts. These findings have been confirmed in larger, more diverse populations.⁵⁻⁷ Prepregnant body mass index (BMI) has been used as the measure of maternal fatness in nearly all of these studies because it is more readily available from medical records than BMI at delivery and is highly associated with maternal fatness at the time when breastfeeding begins.

The means by which being overweight or obese negatively affect lactational performance is unknown and likely to be multifactorial in origin, including the simple mechanical difficulties of latching on and proper positioning of the infant when the mother is obese. In another study in this same geographic area, we observed that prepregnant BMI was negatively associated with the timing of lactogenesis II, the onset of copious milk secretion.⁸ This association was not confounded by the tendency of heavier women to undergo cesarean sections, but it was reduced by primiparity, which was unexpectedly more common among the heavier women in this particular study population. Psychosocial factors, such as planned duration of breastfeeding, behavioral beliefs about breast- and bottle-feeding, and maternal knowledge of and confidence about or support for breastfeeding, were not associated with the timing of the onset of lactogenesis II.

The fall in progesterone concentration that occurs immediately postpartum is the trigger for the onset of copious milk secretion, but maintenance of prolactin and cortisol concentrations is necessary for this trigger to be effective.⁹ It is known that adipose tissue concentrates progesterone.¹⁰ We proposed that this additional source of progesterone, which remains after delivery of the placenta, would lead to consistently higher progesterone concentrations among obese compared with normal-weight women. This, in turn, would lead to a delay in reaching the appropriate concentration to trigger the onset of lactogenesis II. Although the effects of obesity on the prolactin response to infant suckling have never been studied, we postulated that maternal obesity could

compromise this important response. This is because obesity alters the 24-hour spontaneous release of prolactin and also because prolactin secretion is blunted in response to various stimuli among obese subjects.¹¹ Finally, we observed previously that insulin concentrations decrease less dramatically from the end of pregnancy to early lactation in obese than in nonobese rats.¹² We postulated that this may also occur in obese women, which could result in less glucose being available for milk biosynthesis. The goal of the research reported here was to examine whether maternal fatness at the time of conception was associated with several biological factors that are known to be important for the successful initiation of lactation.

METHODS

Pregnant women were identified from the obstetric database at Bassett Healthcare in Cooperstown, New York, and were invited to participate during prenatal visits, during labor, or shortly after delivery. When contact was made before delivery, signed informed consent was returned to study personnel by mail. When contact was made after admission for delivery, signed informed consent was obtained before the first blood sample was obtained. Multiple approaches were used to achieve the desired enrollment, including changing the research protocol to reduce the invasiveness of the procedures and the time and effort required by the participants. We replaced nonmonetary with monetary incentives for the participants because they told us that they would prefer this, and our enrollment rate increased in response. Participants who enrolled after these changes did not differ from those who enrolled earlier. In all, 41 women were eligible for our study and 40 were enrolled during a 24-month period.

At ~48 hours and again at ~7 days postpartum, 3-mL blood samples were drawn from nonfasting participants just before the infant latched on to breastfeed and again 30 minutes later. The time at the beginning of the nursing episode and its duration were recorded. The elapsed time since delivery at the beginning of the nursing episode was calculated. At 48 hours, 40 participants were available to provide data, but blood could be obtained only from 39 of them in the first blood drawing. At 7 days postpartum, 34 participants were available to provide data because 1 had stopped breastfeeding and 5 had been enrolled at a time when the second blood sampling time was at 10 days instead of 7 days postpartum.

Maternal prepregnant weight and height, weight gain during pregnancy, and age as well as newborn weight and length were abstracted from the medical records. Participants were characterized as normal weight when their prepregnant BMI was $<26 \text{ kg/m}^2$ ($n = 23$) and as overweight/obese when their prepregnant BMI exceeded this value ($n = 17$); this choice of cutoff value was based on those used for recommended weight gains during pregnancy by the Institute of Medicine.¹³ Maternal prepregnant BMI was used as a proxy for maternal fatness at this time.

The blood samples were taken in a private setting in the hospital before discharge and again at the time of the return visit at 7 days after birth. Blood sampling began immediately after needle insertion. We had equal success in obtaining blood samples from the 2 groups of participants; only 1 participant required >1 attempt to obtain blood. Sera were separated and frozen at -20°C for later analysis of the concentrations of prolactin, insulin, estradiol, progesterone (all Coat-A-Count kits; Diagnostic Products Corp, Los Angeles, CA, with Lymphochek controls from the same manufacturer), leptin (Human Leptin RIA kit, Linco Research Inc, St Charles, MO, with internal controls), and glucose by standard analytical techniques. The prolactin response to suckling was calculated as the difference between the value after 30 minutes of nursing and the value before nursing began. As recommended by the manufacturers of the kits that we used, data from grossly hemolyzed blood samples (1 at the first drawing on days 7 and 2 at the second drawing on day 7) were excluded from the statistical analyses presented here. Approval for the study was obtained from the University Committee on Human Subjects at Cornell University and the Institutional Review Board at Bassett Healthcare Research Institute.

Statistical Analyses

Normal-weight women were compared with those who were overweight or obese by t test. Data obtained at 48 hours and 7 days postpartum were compared using a paired t test. Associations between pairs of variables were evaluated by univariate regression analysis.

Path analysis was used to develop a parsimonious multivariate prediction of the prolactin response to suckling at 48 hours and 7 days postpartum. As described by Kleinbaum et al,¹⁴ multivariate regression models were built in steps, first evaluating the possibility of an interaction between prepregnant BMI category and the candidate variable on the prolactin response to suckling and then evaluating the possibility of confounding. Candidate variables and their interactions were retained only when they

were statistically significant.

All analyses were conducted with SAS (version 8.2; SAS Institute, Inc, Cary, NC). Data are presented as mean \pm standard deviation. Differences between groups or between observation times were declared to be statistically significant at $P < .05$.

RESULTS

Overweight/obese women gained less weight during pregnancy than the normal-weight comparison group, but, as expected, their BMI at delivery remained significantly higher (Table 1). There were no differences between the groups in maternal age, parity, or the size of the infants at birth.

TABLE 1.

[View inline](#)

Characteristics of Participants and Their Newborn Infants by Maternal Prepregnant BMI Category

The time from delivery to the first measurement was 53.1 ± 15.6 hours ($n = 23$) among the normal-weight women and 51.6 ± 10.2 hours ($n = 17$) among the overweight/obese women; these differences were not significant. For unknown reasons, the time from delivery to the second measurement was significantly ($P < .05$) less among the normal-weight than the overweight/obese women (168.0 ± 30.2 [$n = 16$] vs 196.7 ± 40.4 hours [$n = 17$], respectively). The duration of the nursing episode at the 48-hour measurement did not differ between the groups (~ 21 minutes in both cases), but at 7 days postpartum, the overweight/obese women reported that their infants suckled longer than those of the normal-weight women (23.2 ± 5.6 minutes [$n = 17$] vs 15.3 ± 6.1 minute [$n = 16$], respectively; $P = .0005$).

Prolactin concentrations showed the expected time trend: baseline values decreased significantly from 48 hours to 7 days postpartum in both normal-weight and overweight/obese women. The prolactin response to suckling increased in both groups, significantly ($P < .001$) so in the overweight/obese group (Table 2). The prolactin response to suckling was significantly ($P < .05$) lower in the overweight/obese group than in the normal-weight comparison group at 48 hours but not at 7 days postpartum (Table 2).

TABLE 2.

Prolactin Concentration (ng/mL) Before and After Infant Suckling at 48 Hours and 7 Days Postpartum by Maternal Prepregnant BMI Category

[View inline](#)

Concentrations of both estradiol and progesterone decreased dramatically from 48 hours to 7 days postpartum, as expected (Table 3). There were no significant differences between the groups in estradiol or progesterone values at either time. The decreases in both estradiol and progesterone concentrations from 48 hours to 7 days postpartum did not differ between the prepregnant BMI groups (Table 3).

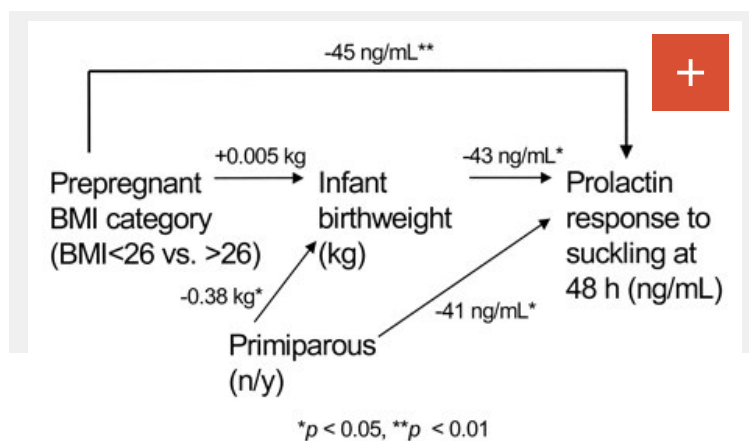
TABLE 3.

[View inline](#)

Metabolic Characteristics and Hormone Concentrations of Participants at 48 Hours and 7 Days Postpartum by Maternal Prepregnant BMI Category

As expected, insulin concentrations decreased from 48 hours to 7 days postpartum; whereas glucose concentrations remained unchanged (Table 3). There were no significant differences between the groups in the concentrations of insulin or glucose at either time, although the insulin values were 30% to 40% higher in the overweight/obese than in normal-weight women. Leptin concentrations did not decrease significantly during the first week postpartum in either group. As expected, leptin concentrations were higher in the overweight/obese group than in the normal-weight group at both times.

In path analyses, overweight/obesity ($P = .006$), primiparity ($P < .02$), and birth weight ($P < .008$) were directly and negatively associated with the increase in prolactin concentration in response to suckling at 48 hours (Fig 1). Compared with normal-weight women, those who were overweight/obese experienced a decrease in the prolactin response to suckling of ~ 45 ng/mL. Compared with multiparous women, those who were primiparous experienced a decrease in the prolactin response to suckling of a similar magnitude. Also, for each addition kilogram of infant birth weight, there was a decrease of a similar magnitude in the prolactin response to suckling.



(n/y)
* $p < 0.05$, ** $p < 0.01$

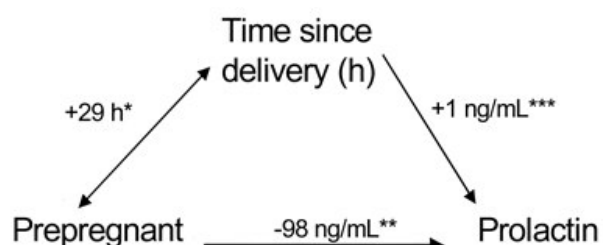
Fig 1.

[Download figure](#) | [Open in new tab](#) | [Download powerpoint](#)

Association of prepregnant BMI category with prolactin response to suckling at 48 hours postpartum. Regression coefficients from path analysis are shown. Statistical significance is indicated. Model for direct effects: adjusted $r^2 = 0.32$, $P < .002$. Total direct and indirect (via birth weight) of prepregnant BMI, -44.0 ng/mL; direct effect of birth weight, -43.2 ng/mL; total direct and indirect (via birth weight) effects of parity, -25.1 ng/mL.

Path analysis also permits the evaluation of indirect pathways. At 48 hours postpartum, there is an indirect pathway from prepregnant BMI category to the prolactin response to suckling via birth weight, which is very small (Fig 1). Thus, the total association of prepregnant BMI via both direct and indirect pathways is ~ -45 ng/mL. There is an indirect pathway of primiparity to the prolactin response to suckling, also via birth weight, which is considerably larger and in the opposite direction of the direct effect. As a result, the total association of primiparity with the prolactin response to suckling via both direct and indirect pathways is ~ -25 ng/mL, or approximately half that of the prepregnant BMI category.

At 7 days postpartum, birth weight and primiparity no longer entered the multivariate regression model; neither did any of the hormone concentrations or changes in the hormone concentrations from 48 hours to 7 days postpartum. Only overweight/obesity remained a significant negative predictor of prolactin response to suckling at 7 days (Fig 2); it was associated with dramatic reduction in the prolactin response to suckling, almost 100 ng/mL ($P < .002$). The results of the path analysis provide an explanation for why the effect of prepregnant BMI category was not evident in the unadjusted analyses. The overweight/obese women had longer breastfeeding episodes, which—as expected—increased their otherwise low prolactin response to suckling. By chance, these women returned for the 7-day visit longer after delivery, which—also as expected—increased their prolactin response to suckling. The indirect pathway from prepregnant BMI to the prolactin response to suckling via duration of the nursing episode was 31 ng/mL and that via time since delivery was 36.5 ng/mL. After accounting for these effects, the total association of prepregnant BMI with the prolactin response to suckling was -31 ng/mL.



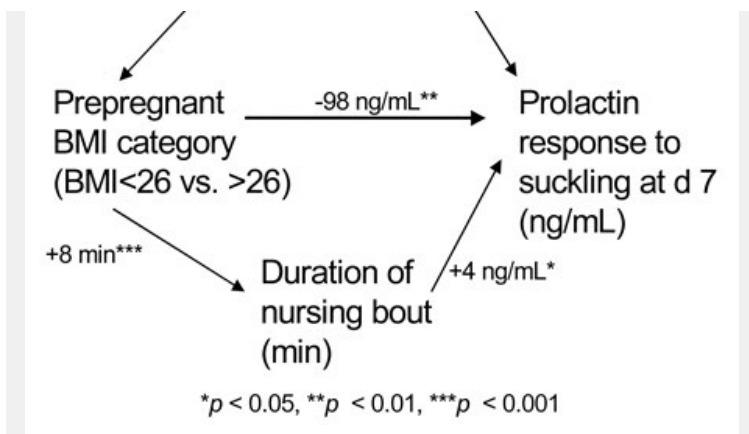


Fig 2.

[Download figure](#) | [Open in new tab](#) | [Download powerpoint](#)

Association of prepregnant BMI category with prolactin response to suckling at 7 days postpartum. Regression coefficients from path analysis are shown. Statistical significance is indicated. Model for direct effects: adjusted $r^2 = 0.47$, $P < .001$. Total of direct and indirect (via time since delivery and duration of nursing) effects of prepregnant BMI, -30.9 ng/mL; direct effect of time since delivery, 1.3 ng/mL; direct effect of duration of nursing episode, 3.9 ng/mL.

DISCUSSION

In this study, we investigated several novel hypotheses. We proposed that the prolactin response to suckling would be reduced in overweight/obese compared with normal-weight women early in the postpartum period. Our hypothesis was confirmed in the unadjusted analyses only at 48 hours postpartum. Through path analysis, we confirmed the importance of being overweight/obese before pregnancy as a predictor of reduced prolactin response to suckling at 48 hours postpartum and, in addition, showed that this effect was of similar magnitude at 7 days postpartum after accounting for confounding factors. These findings are important because, during our observation period (just before and after lactogenesis II, the time of onset of copious milk secretion), the prolactin response to suckling is more important for milk production than it is later in lactation.² We have previously shown that a high proportion of the overweight and obese women in this population who give up on breastfeeding do so at this time.⁴ Thus, although we did not record the duration of breastfeeding in this investigation, there is reason to expect that reduced prolactin response to suckling in the early postpartum period would be associated with shortened duration of breastfeeding and thus would represent 1 means by which excess maternal weight could contribute to poor lactational performance.

We chose to measure prolactin response to suckling at 30 minutes after the beginning of the suckling episode on the basis of reports in the

literature that this was the time of maximum response.^{15,16} The values that we observed for baseline prolactin as well as the prolactin response to suckling among normal-weight women at 7 days postpartum were very similar to those at 10 days postpartum reported by Battin et al.¹⁶

We postulated that there would be consistently higher progesterone concentrations in the early postpartum period among obese compared with normal-weight women because adipose tissue is an extraplacental source of this hormone. This hypothesis was not supported in this study because there were no significant differences between normal-weight and overweight/obese women in progesterone concentrations at either 48 hours or 7 days postpartum. The values that we observed at these times were similar to those reported by others in the early postpartum period.¹⁷⁻¹⁹

In addition, we examined the association between maternal overweight/obesity and the decrease in estrogen concentration that occurs postpartum. We were interested in this possible association because estradiol regulates the transcription of the prolactin gene²⁰ and is another hormone with an extraplacental source in obese individuals. As expected, estradiol concentrations decreased significantly during the observation period in both groups of women. Our values were similar to those reported previously by others for normal-weight women in the early postpartum period.^{17,19,21} Neither the concentration of estradiol nor the change in estradiol during the observation period was related to the prolactin response to suckling. The high estradiol concentrations that are characteristic of pregnancy stimulate the development of the mammary gland during lactogenesis I, but estradiol is not among the hormones required for lactogenesis II, the onset of copious milk secretion, or lactogenesis III, the maintenance of established lactation.²

In the first few days postpartum, insulin concentrations fall from the high values that are characteristic of late pregnancy to concentrations similar to those of nonpregnant women.²² On the basis of our previous research in rats,¹² we proposed that this decrease in insulin concentration from pregnancy to lactation would be compromised in overweight/obese women. Blood samples were not obtained in late pregnancy, but there was a decrease in insulin concentration between 48 hours and 7 days postpartum. This decrease was statistically significant among the normal-weight women and approached significance ($P < .06$) among the overweight/obese women. In addition, insulin concentrations were 28% and 44% higher among the obese/overweight women than the normal-

weight women at 48 hours and 7 days postpartum, respectively, but these differences were not statistically significant. As we were unable to obtain fasting blood samples in this investigation, our measures of insulin have greater variability than in fasting samples, and this likely reduced our ability to detect the differences that we postulated would be present.

Leptin concentrations rise linearly with BMI,²³ so the higher leptin values that we observed among the overweight/obese compared with the normal-weight women were expected. The difference between normal-weight and overweight/obese women was greater at 48 hours than at 7 days. Leptin values rise during pregnancy and return to nonpregnant values within a few days after delivery.^{21,23,24} Leptin concentrations decreased in both groups during the observation period, but in neither group was this decrease statistically significant. Butte et al²⁵ observed an inverse association between leptin and prolactin concentrations at 3 and 6 months postpartum. In the present investigation, leptin concentration was not associated with baseline prolactin concentration at either 48 hours or 7 days postpartum. In contrast, leptin was negatively associated ($P < .03$) with the prolactin response to suckling at 7 days postpartum only. Leptin was not statistically significant when added to the path analysis model shown in Fig 2. This indicates that leptin concentration does not provide additional information about the regulation of the prolactin response to suckling beyond that contained in the prepregnant BMI category.

Although the population served by Bassett Healthcare has participated willingly in previous survey-based research, this was not the case in the present study. This is perhaps not surprising because these participants were asked for blood samples at a very sensitive time, before and after nursing twice during the first week postpartum. In addition, they were asked to make other measurements (eg, an infant milk intake, which required weighing the infant before and after each feed during two 24-hour periods) that were perceived as burdensome. This compromised our ability to collect all of the data that would have been desirable in this investigation and also our ability to do so under optimal conditions for sample analysis (eg, fasting conditions).

The unique and important finding from this study is that overweight/obese women had a lower prolactin response to suckling. This would be expected to compromise the ability of overweight/obese women to produce milk and, over time, could lead to premature cessation of lactation, a phenomenon that we and others have previously observed in women with a high prepregnant BMI in this and other populations. This

finding thus provides evidence of a biological basis for this association, and additional study of it is likely to be informative. The data did not support our hypothesis of higher progesterone or estradiol concentrations in overweight/obese compared with normal-weight women that might delay lactogenesis. The data also did not support our hypothesis of a difference in the pattern of change in insulin values during the early postpartum period. Inasmuch as the postulated trends were present, the lack of statistical significance may have been attributable to the use of nonfasting samples or inadequate statistical power.

The findings from this study add plausibility to our observation that initiation, not just duration of breastfeeding, was negatively affected by maternal overweight/obesity.⁸ Although women should begin pregnancy at a healthy weight and gain reasonably during gestation, not all will. Pediatricians can help overweight/obese women to succeed at breastfeeding by targeting them for contact with a lactation consultant before discharge from the hospital to be sure that they have received optimal advice on breastfeeding techniques. In addition, early contact with the mother after discharge—by calling her at home to offer her support and counseling for breastfeeding, by scheduling the first pediatric visit earlier than for other patients, or by enlisting the assistance of public health nurses for a home visit if this is possible—would help overweight/obese women to continue to breastfeed.

Acknowledgments

This research was supported by a grant from the US Department of Agriculture (NRI 9900814).

We thank the nurses at the Bassett Healthcare Birthing Center, Leslie Davidson and Chris Burrington for performing the blood determinations, and Rob Anderson for assistance with data management.

Footnotes

- Received July 24, 2003.
- Accepted December 15, 2003.
- Address correspondence to Kathleen M. Rasmussen, ScM, ScD, RD, Division of Nutritional Sciences, 111 Savage Hall, Cornell University, Ithaca, NY 14853-6301. E-mail: kmr5@cornell.edu
BMI, body mass index

REFERENCES

1. ↗ US Department of Health and Human Services. *Healthy People 2010*. Washington, DC: Department of Health and Human Services; 2000
2. ↗ Lawrence RA, Lawrence RM. *Breastfeeding: A Guide for the Medical Profession*. St. Louis, MO: Mosby; 1999
3. ↗ Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999-2000. *JAMA*. 2002;288 :1723– 1727
4. ↗ Hilson JA, Rasmussen KM, Kjolhede CL. Maternal obesity and breastfeeding success in a rural population of Caucasian women. *Am J Clin Nutr*. 1997;66 :1371– 1378
5. ↗ Donath SM, Amir LH. Does maternal obesity adversely affect breastfeeding initiation and duration? *J Paediatr Child Health*. 2000;36 :482– 486
6. Li R, Ogden C, Ballew C, Gillespie C, Grummer-Strawn LM. Prevalence of exclusive breastfeeding among US infants: the Third National Health and Nutrition Examination Survey (Phase II, 1991-1994). *Am J Public Health*. 2002;92 :1107– 1110
7. ↗ Sebire NJ, Jolly M, Harris JP, et al. Maternal obesity and pregnancy outcome: a study of 287 213 pregnancies in London. *Int J Obes*. 2001;25 :1175– 1182
8. ↗ Hilson JA, Rasmussen KM, Kjolhede CL. High prepregnant body mass index is associated with poor lactation outcomes among white, rural women independent of psychosocial and demographic correlates. *J Hum Lact*. 2004;20 :18– 29
9. ↗ Neville MC, Morton S. Physiology and endocrine changes underlying human lactogenesis II. *J Nutr*. 2001;131 :3005S– 3008S
10. ↗ Azziz R. Reproductive endocrinologic alterations in female asymptomatic obesity. *Fertil Steril*. 1989;52 :703– 725
11. ↗ Kopelman PG. Physiopathology of prolactin secretion in obesity. *Int J Obes*. 2000;24 (suppl 2):S104– S108
12. ↗ Shaw MA, Rasmussen KM, Myers TR. Consumption of a high-fat diet impairs reproductive performance in Sprague-Dawley rats. *J Nutr*. 1997;127 :64– 69
13. ↗ Institute of Medicine (Subcommittees on Nutritional Status and Weight Gain During Pregnancy and Dietary Intake and Nutrient Supplements During Pregnancy, Committee on Nutritional Status During Pregnancy and Lactation, Food and Nutrition Board). *Nutrition During Pregnancy: Part I, Weight Gain; Part II, Nutrient Supplements*. Washington, DC: National Academy Press; 1990
14. ↗ Kleinbaum DG, Kupper LL, Muller KE, Nizam A. *Applied Regression Analysis and Other Multivariable Methods*. Pacific Grove, CA: Duxbury Press; 1998
15. ↗ 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

16. ↗ Battin DA, Marrs RP, Fleiss PM, Mishell DR Jr. Effect of suckling on serum prolactin, luteinizing hormone, follicle-stimulating hormone, and estradiol during prolonged lactation. *Obstet Gynecol.* 1985;65 :785– 788
17. ↗ Martin RH, Glass MR, Chapman C, Wilson GD, Woods KL. Human alpha-lactalbumin and hormonal factors in pregnancy and lactation. *Clin Endocrinol.* 1980;13 :223– 230
18. Dawood MY, Khan-Dawood FS, Wahi RS, Fuchs F. Oxytocin release and plasma anterior pituitary and gonadal hormones in women during lactation. *J Clin Endocrinol Metab.* 1981;52 :678– 683
19. ↗ Ingram JC, Woolridge MW, Greenwood RJ, McGrath L. Maternal predictors of early breast milk output. *Acta Paediatr.* 1999;88 :493– 499
20. ↗ Maxwell CA. Estradiol regulates the transcription of the prolactin gene. *J Biol Chem.* 1982;257 :2133– 2136
21. ↗ Schubring C, Englaro P, Siebler T, et al. Longitudinal analysis of maternal serum leptin levels during pregnancy, at birth, and up to six weeks after birth: relation to body mass index, skinfolds, sex steroids and umbilical cord blood leptin levels. *Horm Res.* 1998;50 :276– 283
22. ↗ Lind T, Harris VG. Changes in the oral glucose tolerance test during the puerperium. *Br J Obstet Gynaecol.* 1976;83 :460– 463
23. ↗ Hardie L, Trayhurn P, Abramovich P, Fowler P. Circulating leptin in women: a longitudinal study in the menstrual cycle and during pregnancy. *Clin Endocrinol.* 1997;47 :101– 106
24. ↗ Mukherjea R, Castonguay TW, Douglass LW, Moser-Veillon P. Elevated leptin concentrations in pregnancy and lactation: possible role as a modulator of substrate utilization. *Life Sci.* 1999;65 :1183– 1193
25. ↗ Butte NF, Hopkinson JM, Nicolson MA.. Leptin in human reproduction: serum leptin levels in pregnant and lactating women. *J Clin Endocrinol Metab.* 1997;82 :585– 589

- Copyright © 2004 by the American Academy of Pediatrics

[View Abstract](#)



Previous

Next

