Lactation After Normal Pregnancy Is Not Associated With Blood Glucose Fluctuations

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Preastfeeding is the preferred method of feeding infants up to 12 months of age (1). Breastfed infants experience fewer and less severe infections and may be protected against future disease development (2). Mothers who breastfeed potentially experience accelerated weight loss (3), a lower risk of breast and ovarian cancer (3), and a lower risk of type 2 diabetes than mothers who do not breastfeed (4).

Breastfeeding is recommended for all women, including those with gestational (5) or pregestational (6) diabetes. However, data suggest that diabetic women may experience hypoglycemia during breastfeeding; subsequently, they have been advised to eat before or during breastfeeding to avoid hypoglycemia (6). Because data on blood glucose fluctuations in normoglycemic women are limited, we tested the hypothesis that lactation in healthy normoglycemic women will not cause significant blood glucose fluctuations.

RESEARCH DESIGN AND

METHODS — Women were recruited randomly from the Pregnancy Program at Brigham and Women's Hospital (BWH). The BWH Human Research Committee approved the study protocol, and all women provided informed written consent before participation. Healthy women were included if they were 18–40 years of

age, 6 weeks to 6 months postpartum, free of a history of gestational or pregestational diabetes, and breastfeeding with no more than one bottle per day of supplementation. Women were excluded if they had an abnormal 1-h glucose challenge test during pregnancy (5), were taking medications known to influence blood glucose, were nursing more than one infant, or were incapable of using the continuous glucose monitoring system (CGMS).

Participants presented to the general clinical research center (GCRC) of BWH, where height and weight were measured and pregnancy history reviewed. A study physician inserted the CGMS according to manufacturer directions and provided participants with instructions regarding CGMS maintenance and Accu-Chek finger-stick blood glucose monitoring for CGMS calibration.

Participants wore the CGMS for 48 h and documented initiation of infant suckling or breast pump use, food intake, finger-stick blood glucose monitoring (at least four times daily), hypoglycemic symptoms, exercise, and sleep. In addition, participants marked these events using CGMS controls. After 48 h, participants returned to the GCRC and had fasting blood drawn for glucose, insulin, and A1C. The physician collected the documentation, removed the CGMS, and downloaded the CGMS data.

Blood samples were iced and centrifuged at 4°C and 2,000 rpm, respectively, for 15 min. Plasma was frozen at -70°C until assays were performed. Serum glucose was measured by the glucose oxidase method (Beckman glucose analyzer; Beckman, Fullerton, CA). Serum insulin was assayed by chemiluminesence (Beckman Chemiluminesence new protocol; Beckman, Chaska, MN). A1C was measured by high-performance liquid chromatography (Tosoh Bioscience, San Francisco, CA).

To optimize data accuracy by allowing for CGMS acclimation, blood glucose levels obtained during the second 24-h CGMS period were analyzed. The first lactation episode free of food or caloric beverage intake for 60 min both before and after lactation initiation was identified, and data were analyzed using SAS (version 9.1; SAS Institute, Cary, NC). ANOVA was used to compare the glucose levels obtained before and after lactation. Paired *t* tests were used to compare 1) the mean area under the curve for glucose (AUCg) before lactation to the mean AUCg after lactation and 2) the mean glucose before to the mean glucose after lactation. Data are expressed as means \pm SD, and P < 0.05 was considered statistically significant.

RESULTS — The nine women studied were 33 \pm 4 years of age and 20.3 \pm 7.3 weeks postpartum. They had BMI 24.4 ± 5.3 kg/m^2 , fasting glucose $82 \pm 8 \text{ mg/dl}$, insulin 1.99 \pm 0.90 μ U/ml, and A1C $5.5 \pm 0.3\%$. The CGMS data revealed no significant fluctuations in glucose levels obtained at 5-min intervals during the prelactation period (P = 0.23) (Fig. 1). Similarly, there were no significant fluctuations among the glucose levels obtained during the period after lactation (P = 0.09). The mean glucose level before lactation did not differ significantly compared with the mean glucose level after lactation (95 \pm 14 vs. 96 \pm 15 mg/dl; P =0.93). The mean AUCg before lactation did not differ significantly compared with the mean AUCg after lactation (376.9 ± $53.3 \text{ vs. } 382.8 \pm 56.9 \text{ mg} \cdot \text{h}^{-1} \cdot \text{dl}^{-1}; P =$ 0.34).

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Published ahead of print at http://care.diabetesjournals.org on 13 August 2007. DOI: 10.2337/dc07-1243. **Abbreviations:** AUCg, area under the curve for glucose; BWH, Brigham and Women's Hospital; CGMS,

continuous glucose monitoring system; GCRC, general clinical research center.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

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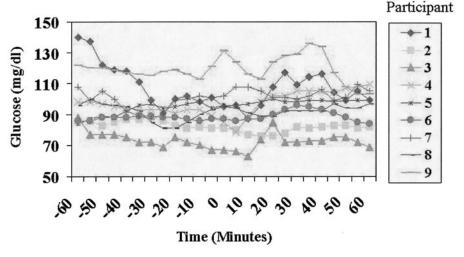


Figure 1— Blood glucose levels (mg/dl) collected every 5 min during the 60 min before and after the start of lactation (time = 0) for each of the nine participants in the study.

CONCLUSIONS — We observed no significant glucose changes during the period before or after lactation in women who had pregnancies uncomplicated by pregestational or gestational diabetes. Moreover, these healthy women did not have differences in the mean glucose levels obtained prelactation compared with postlactation, nor did they manifest hypoglycemia in response to lactation.

Our data extend the current literature on blood glucose response to lactation by examining healthy women. Data in type 1 diabetic women have revealed that breast-feeding lowered blood glucose concentrations and made achieving metabolic control more difficult (6). Studies have demonstrated that women with gestational diabetes benefit from lactation, manifesting improved glucose metabolism with a twofold increase in subsequent type 2 diabetes among nonlactating women (7). However, to our knowledge, there are no published data using CGMS

to characterize glucose fluctuations in healthy lactating women.

Our main study limitation is sample size. However, although only nine women were studied, each had CGMS data providing blood glucose sampling every 5 min both before and after lactation to understand blood glucose characteristics during this period. Additionally, the validity of our selected blood glucose analysis periods before and after lactation was dependent on participant reliability in documenting food intake.

In conclusion, we observed no significant blood glucose fluctuations in response to lactation among healthy women with a normoglycemic pregnancy history. Therefore, these women do not need to increase caloric intake specifically before or during lactation to prevent hypoglycemia. This is noteworthy because limiting postpartum caloric intake will facilitate the loss of excess pregnancy weight, thereby reducing the risk of developing

obesity. Nonetheless, women should be aware of recommendations regarding healthy caloric intake during breast feeding (8). Moreover, further studies of glucose fluctuations during lactation in larger numbers of normoglycemic and diabetic women are warranted.

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