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Obesity as a Predictor of Delayed Lactogenesis II

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Abstract

Background: Lactogenesis II is the onset of copious milk production. A delay in this has been associated with an increased risk of formula supplementation and early cessation of breastfeeding. Prepregnancy obesity has also been associated with decreased breastfeeding rates and early cessation.

Research aim: This study aimed to evaluate the effect of prepregnancy obesity on self-reported delayed lactogenesis II.

Methods: We conducted a prospective observational cohort study of 216 women with a singleton pregnancy and who planned to breastfeed. We compared the onset of lactogenesis II between women with a body mass index (BMI) < 30 kg/m^2 and women with a BMI ≥ 30 kg/m^2. Using multivariate logistic regression analyses, we assessed the relationship between maternal BMI and delay of lactogenesis II.

Results: The prevalence of delayed lactogenesis II among women with prepregnancy BMI < 30 kg/m^2 and BMI ≥ 30 kg/m^2 was 46.4% and 57.9%, respectively. Delayed lactogenesis II occurred more frequently among women who were obese at the time of delivery ($p < .05$). After controlling for the covariates, age, prepregnancy BMI, and gestational weight gain were positively associated with delayed lactogenesis II.

Conclusion: Prepregnancy obesity and excessive gestational weight gain are associated with an increased risk of delayed lactogenesis II. Women who are at risk for delay in lactogenesis II and early breastfeeding cessation will need targeted interventions and support for them to achieve their personal breastfeeding goals.

Keywords

breastfeeding, breastfeeding duration, breastfeeding initiation, breastfeeding rates, lactation, lactogenesis

Background

It is widely acknowledged that breastfeeding is the choice of feeding for all infants (Ip et al., 2007; Kramer & Kakuma, 2012; World Health Organization, 2014). The World Health Organization recommends exclusive breastfeeding for the first 6 months of life, followed by the introduction of solids and continued breastfeeding into the 2nd year and beyond.

The onset of milk secretion is called lactogenesis, a process that is divided into two stages (Neville, 2013). Lactogenesis I includes the stage of secretory differentiation, which starts at 16 weeks gestation. Lactogenesis II occurs after birth and is associated with copious milk secretion. At the onset of lactogenesis II, women report a sense of fullness and swelling of the breasts and may report leakage of milk. The mother typically reports symptoms between 50 and 72 hr postpartum, and lactogenesis II is often referred to as "milk coming in."

The onset of lactogenesis II after 72 hr postpartum is defined as delayed lactogenesis II (DLII; Brownell, Howard, Lawrence, & Dozier, 2012; Matias, Dewey, Quesenberry, & Gunderson, 2014). The incidence of DLII ranges from 17% to 44% (Brownell et al., 2012; Chapman & Pérez-Escamilla, 1999a). Infants of mothers who experience DLII experience excessive weight loss and therefore are at high risk for formula supplementation even if the mother's goal is to exclusively breastfeed. Furthermore,

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women who experience DLII are at high risk for early cessation of breastfeeding (Chapman & Pérez-Escamilla, 1999b; Nommsen-Rivers, 2016). Studies have shown that a high body mass index (BMI) is associated with DLII (Matias et al., 2014; Turcksin, Bel, Galjaard, & Devlieger, 2014). For instance, in a cohort of 877 women with gestational diabetes during pregnancy, 33% of women reported DLII, and it was associated with prepregnancy obesity (odds ratio [*OR*] = 1.56; 95% confidence interval [CI] [1.07, 2.29]). Another potential link to obesity and delayed lactogenesis is the risk of excessive weight gain in pregnancy. Bartok, Schaefer, Beiler, and Paul (2012) conducted a prospective longitudinal cohort study of 718 postpartum women. Although they did find an association between gestational weight gain and DLII, this relationship did not persist after controlling for covariates. Other maternal factors that are associated with a later onset of lactogenesis II include increasing maternal age, primiparity, gestational diabetes, operative delivery, labor pain medication, and nipple pain when breastfeeding (García-Fortea et al., 2014; Lind, Perrine, & Li, 2014; Prior et al., 2012). Neonatal factors that are associated with the onset of lactogenesis II are birth weight, supplementation within 48 hr postdelivery, and excess neonatal weight loss (Brownell et al., 2012; Chantry, Dewey, Peerson, Wagner, & Nommsen-Rivers, 2014; Moore, Anderson, Bergman, & Dowswell, 2012).

Women who are obese (defined as a BMI ≥ 30 kg/m^2) prior to pregnancy are less likely to initiate breastfeeding and more likely to have shorter duration of breastfeeding (Babendure, Reifsnider, Mendias, Moramarco, & Davila, 2015; Wojicki, 2011). They are also more likely to discontinue breastfeeding due to a need for formula supplementation and are less likely to receive breastfeeding support during hospitalization (Kair & Colaizy, 2016a, 2016b). More than one third of adults in the United States are obese (Centers for Disease Control and Prevention, 2015). Because women who are overweight or obese are also more likely to have comorbidities such as preexisting diabetes mellitus and gestational diabetes mellitus and are at higher risk for cesarean section and macrosomic infant birth, these factors may influence breastfeeding outcomes (Marchi, Berg, Dencker, Olander, & Begley, 2015).

Given this high prevalence and the fact that overweight and obese women experience more difficulties in breastfeeding, it is extremely important to obtain a better understanding of those factors predictive of successful breastfeeding outcomes. The study aim was to investigate the effect of prepregnancy obesity on the rates of DLII.

Key Messages

- Little is known about what factors contribute to decreased breastfeeding rates among women with obesity.
- Women with prepregnancy obesity were more likely to experience delayed lactogenesis II.
- Women with high prepregnancy body mass index and excessive gestational weight gain should be offered tailored lactation support.
- Further research is needed to better understand the physiologic reason for breastfeeding difficulties in women with prepregnancy obesity.

Methods

Design

This prospective observational cohort study was performed at a single urban tertiary care center. The study protocol was approved by the institutional review board of the University of South Florida.

Setting

Tampa General Hospital is a 1,021-bed academic tertiary medical center that is Baby-Friendly designated (Baby-Friendly USA, 2012). It is located in the southeastern United States and serves a diverse patient population, and the primary payor is Medicaid. Locally, 77% of newborns are ever breastfed (Centers for Disease Control and Prevention, 2014).

Sample and Data Collection

We prospectively enrolled a convenience sample of women with and without obesity into a protocol to examine the association of prepregnancy obesity on DLII. Potential participants were approached in the examination room during a scheduled prenatal visit between 34 and 37 weeks' gestation by a research team member. Recruitment flyers were posted in the clinic space and women were also able to call the research team for enrollment. Women were included if they were 18 years or older, were expecting a live-born infant, were carrying a singleton pregnancy, expressed the desire/motivation to breastfeed, and spoke the English language fluently. Women were excluded if they were carrying a multiple gestation, had a history of breast reduction or enlargement surgery, or had no telephone access. A second screening occurred after delivery. Participants who delivered preterm (< 37 weeks of gestation), mothers who were not admitted to the mother–infant unit (i.e., admitted to intensive care or special care unit or labor & delivery unit for postpartum monitoring), and mothers who did not initiate breastfeeding within 12 hr after delivery were excluded from further analysis.

The study was powered to answer the research question, What is the impact of obesity on DLII? We assumed enrollment of women who were obese (BMI ≥ 30 kg/m^2) to women who were not obese (BMI < 30 kg/m^2), based on prepregnancy weight, in a 1:1 fashion. Assuming a rate of 30% DLII

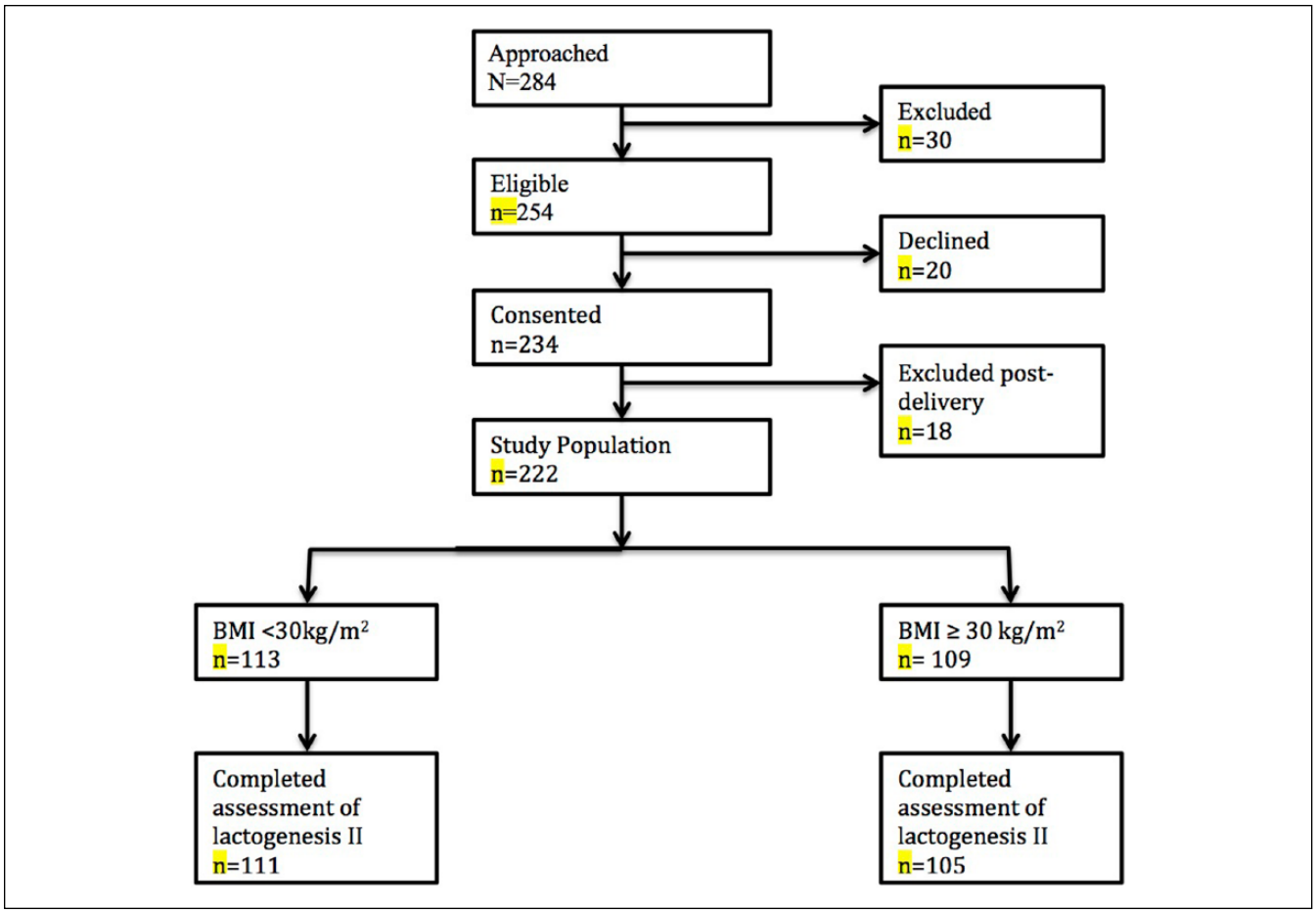

Figure 1. Enrollment of participants.

among the overall population, to detect an absolute difference of 20% in the obese group, we would need 93 women in each group (obese vs. normal weight) to detect a difference with 80% power and $\alpha = .05$. Anticipating an approximate dropout/lost to follow-up rate of 30%, we estimated a priori that we would need to approach up to 241 women.

Between August 27, 2014, and October 15, 2015, we enrolled 216 participants in the study. Initially, the obesity rate of the women who were enrolled was lower than expected, leading to the inclusion of more women with a BMI < 30 kg/m^2 than expected to reach an equal number of women in each BMI group; we had to specifically target women with a BMI ≥ 30 kg/m^2 toward the end of the study period. In the end, we approached 284 women (see Figure 1). Of those women, 254 met the inclusion criteria, and 234 agreed to participate and signed the informed consent. Twenty women declined to participate. The reasons for the postdelivery exclusions were as follows: 9 women did not meet the inclusion criteria after delivery (failed to breastfeed $n = 2$, infant in neonatal intensive care unit $n = 3$, preterm delivery $n = 3$, delivered at another hospital $n = 1$). One participant had withdrawn without providing a reason, and an additional 8 women were lost to follow-up postdelivery before we assessed the onset of lactogenesis II. Ultimately, 216 women met the inclusion criteria after delivery and completed the assessment of lactogenesis II follow-up.

Ethics

Informed consent was obtained in a private room. Potential risks to the patient included a breach of confidentiality and coercion to breastfeed. All questioning regarding lactation and breastfeeding was conducted in a private setting to ensure maximum confidentiality.

Measurement

Prenatally, women completed the Infant Feeding Intentions scale (Nommsen-Rivers & Dewey, 2009). This five-item questionnaire uses a 5-point Likert-type scale format (0-4) to inquire regarding level of agreement with five infant feeding statements (see Table 1). The first two items measure intent to initiate breastfeeding. The following items assess strength of intent to provide exclusive human milk at 1, 3, and 6

Table 1. Maternal Demographics and Covariates by Delayed Lactogenesis II (DLII) Status.

Characteristics	*n* (%) or mean		*p*
	DLII no (*n* = 105)	DLII yes (*n* = 114)	
Demographics			
Age (years)	28	30.5	.10
Hispanic	17 (16.3)	22 (19.3)	.57
Race			
White	76 (73.1)	89 (78.1)	
Black or African American	19 (18.3)	20 (17.5)	
Asian	3 (2.9)	2 (1.8)	
Pacific Islander	1 (1.0)	1 (0.9)	
Other	5 (4.8)	2 (1.8)	.74
Prenatal care provider			
Midwife group	29 (27.6)	33 (29.2)	
Staff	49 (46.7)	55 (48.7)	
Faculty	27 (25.7)	25 (22.1)	.82
Maternal factors			
Prepregnancy obesity	45 (42.9)	62 (54.4)	.08
Time of delivery obesity	61 (58.1)	83 (72.8)	.02
Excessive weight gain[a]	44 (41.9)	59 (51.8)	.14
Primipara	42 (40.0)	54 (47.4)	.27
Tobacco use during pregnancy	8 (7.6)	7 (6.1)	.66
Marijuana use during pregnancy	4 (3.8)	2 (1.8)	.43
Opioid use during pregnancy[b]	1 (1.0)	0 (0.0)	.47
Diabetes			
Type 1	0 (0.0)	1 (0.5)	
Type 2	2 (1.9)	6 (3.7)	
Gestational diabetes	8 (7.6)	10 (8.2)	.415
Hypertension			
Chronic	10 (9.5)	10 (8.8)	
Gestational hypertension	6 (5.7)	6 (5.3)	
Preeclampsia	2 (1.9)	5 (4.4)	.805
Hypothyroidism	8 (7.6)	7 (6.1)	.665
Asthma	16 (15.2)	23 (20.2)	.340
Symptoms of depression[c]	19 (18.1)	20 (17.5)	.915
Labor and delivery			
Cesarean section	41 (39.0)	48 (42.1)	.645
Epidural anesthesia	38 (59.4)	55 (83.3)	.002
Oxytocin use	52 (49.5)	67 (58.8)	.170

[a]According to the recommendations of the Institute of Medicine guidelines for weight gain in pregnancy. [b] Methadone maintenance or urine drug screen positive for opioids. [c] Total score ≥ 9 on the Edinburgh Postnatal Depression Scale.

months of age. The questionnaire has a maximum score of 20, meaning full intention to exclusively breastfeed for at least 6 months. The original validation study included a diverse group of women (n = 170) and demonstrated adequate reliability (Cronbach's alpha coefficient = .90).

After the participant had delivered in the hospital, she was visited or called between 12 and 36 hr postpartum for a baseline assessment. At this visit, we determined the postdelivery eligibility. If the participant still met the inclusion criteria, we inquired about her breastfeeding experience thus far. We asked the participant about the timing of the onset of milk coming in by using the Assessment of Onset of Lactogenesis II (Chapman & Pérez-Escamilla, 2000). Maternal perception of onset of lactation has been demonstrated to be a valid indicator of onset of lactogenesis II when compared with the gold standard of test weights; the sensitivity and specificity were 71.4% and 79.3%, respectively (Chapman & Pérez-Escamilla, 2000). Delayed lactogenesis II was defined as an onset of lactogenesis II after 72 hr postpartum (Brownell et al., 2012; Matias et al., 2014).

LATCH is a breastfeeding charting system that provides a systematic method for gathering information about individual breastfeeding sessions (Jensen, Wallace, & Kelsay, 1994). The LATCH score is documented every 8 hr by staff

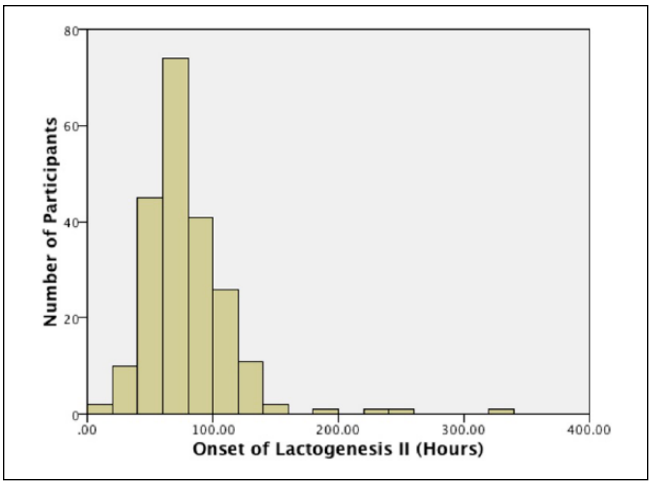

Figure 2. Onset of lactogenesis II among study population.

nurses trained in breastfeeding assessment and included as a part of the medical record. A low LATCH score within 24 hr of delivery has been associated with a high risk of formula supplementation before hospital discharge (Tornese et al., 2012). The LATCH score closest to 24 hr postpartum was used for the data analysis.

The covariates analyzed in this study included maternal, infant, and birth-related factors known to be associated with breastfeeding outcomes. Maternal factors such as age, parity, obesity prepregnancy and at the time of birth, excessive weight gain during pregnancy, (gestational) diabetes, maternal smoking, and maternal endocrine disorders (Finkelstein et al., 2013; Kitano et al., 2016; Ogbo et al., 2017; Turcksin et al., 2014). Delivery factors collected included mode of birth (spontaneous vaginal birth, instrumental vaginal birth, cesarean section), labor pain medication, and oxytocin use (García-Fortea et al., 2014; Lind et al., 2014; Prior et al., 2012). Infant and feeding factors included birth weight, 1- and 5-min Apgar scores, skin-to-skin contact during the 1st hr, time to first feeding at breast, LATCH score, type of nipples, any neonatal formula supplementation prior to discharge, and documented nipple pain when breastfeeding (Chantry et al., 2014; Moore et al., 2012; Tornese et al., 2012).

All the covariates concerning delivery and postdelivery were abstracted from the participant's medical chart as documented by the clinician or nurse. Prepregnancy BMI was calculated as maternal prepregnancy weight/maternal height, found in the women's medical record. BMI at delivery was calculated using the maternal weight at last prenatal visit and the documented height. Women with a BMI ≥ 30 kg/m^2 were categorized as obese (Centers for Disease Control and Prevention, 2015). Gestational weight gain was calculated as the difference between the prepregnancy weight and the weight at the last prenatal visit. We classified excess gestational weight gain according to the recommendations of the American College of Obstetricians and Gynecologists (2013).

Data Analysis

Analyses were performed to measure mothers who had and had not experienced DLII across identified maternal demographic characteristics, obstetric factors, obesity, and various lifestyle factors. Continuous variables were analyzed with either the Student t test or Mann-Whitney U test if nonparametric. Categorical variables were analyzed with χ^2 or Fisher exact tests where appropriate. For multivariate analyses, all variables of interest were included in a logistic regression model. Logistic regression was used to control for covariates. A backward stepwise approach utilizing the likelihood ratio test to assess the impact of each covariate on the model was used. Variables were included in the model if they were significant at $p < .10$ in the univariate analysis or were known covariate variables. The variables included in the regression analysis were age, race, ethnicity, prepregnancy BMI, delivery BMI, gestational weight gain, cesarean delivery, epidural, and time from delivery to infant at breast. Analyses were conducted in SPSS Statistics Version 23.0 (IBM Corporation, Armonk, NY).

Results

Women were contacted for up to 14 days to determine the onset of DLII. The self-reported, maternal perception of onset of lactogenesis II ranged from 11 to 336 hr postpartum, with a median of 73 hr (see Figure 2). One woman had an onset of lactogenesis II at 336 hr. Among the women with a prepregnancy BMI < 30 kg/m^2, 46.4% experienced a delay in lactogenesis II (> 72 hr) as compared with 57.9% of women with a BMI ≥ 30 kg/m^2 ($p = .088$).

The maternal demographic and clinical characteristics of the cohort are demonstrated in Table 1 by DLII status, and the infant and breastfeeding factors are demonstrated in Table 2. Maternal prepregnancy BMI ranged from 15.6 to 63.8 kg/m^2, with a mean (standard deviation) of 31.3 (9.6) kg/m^2. The mean (standard deviation) BMI in the nonobese group was 23.9 (3.2) kg/m^2 and in the obese group, 38.9 (7.9) kg/m^2.

Women who experienced delayed lactogenesis were similar in age, race, and ethnicity to women who did not experience DLII. Women with DLII were more likely to be obese at delivery but had similar gestational weight gain. Epidural use was high among women with DLII but oxytocin use was similar. The women with DLII were also less likely to have had the infant at breast within the 1st hr after delivery. There was a positive correlation between the number of hr from delivery to onset of lactogenesis II and prepregnancy BMI ($r = .216$, $p < .01$) and delivery BMI ($r = .289$, $p < .01$) (see Figure 3). After controlling for covariates, increasing age, prepregnancy BMI, and gestational weight gain were associated with DLII (see Table 3).

Discussion

In our study, we found that delayed lactogenesis was more prevalent among women who were obese prepregnancy, with

Table 2. Infant and Feeding Characteristics by Delayed Lactogenesis II (DLII) Status.

Characteristics	*n* (%) or mean		*p*
	DLII no (*n* = 105)	DLII yes (*n* = 114)	
Birth weight (g)	3,464	3,535	.35
Apgar 1 min < 8	4 (5.9)	3 (4.6)	> .99
Apgar 5 min < 8	0 (0.0)	2 (3.1)	.23
Skin-to-skin contact within 1 hr	79 (75.2)	87 (80.6)	.34
Breastfeeding characteristics			
At breast < 1 hr	57 (60.0)	58 (44.0)	.02
Latch score < 8	23 (23.2)	28 (25.7)	.68
Supplement of formula < 48 hr	42 (40.0)	55 (48.2)	.22
Flat/inverted nipple[a]	3 (2.9)	10 (8.9)	.06
Nipple pain during breastfeeding	14 (13.6)	15 (13.4)	.96

[a]Observed by nurse as part of the LATCH score.

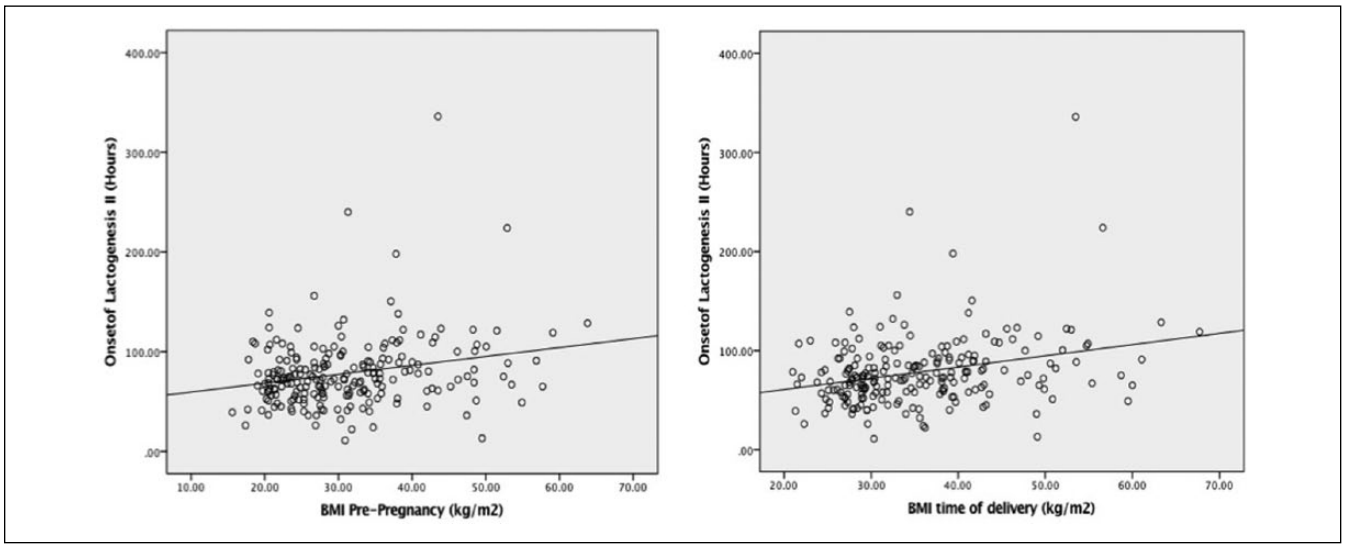

Figure 3. Correlation between maternal (prepregnancy and delivery) body mass index and the onset of lactogenesis II (both *p*s < .01).

an absolute increase of 11.5%. Our findings are consistent with prior published studies (Matias et al., 2014; Turcksin et al., 2014). Furthermore, our study suggests that excessive gestational weight gain was also associated with a delay in lactogenesis II. If our finding is confirmed, this is a potential target for improving onset of lactogenesis II and subsequent exclusive breastfeeding rates.

It is interesting to note that the mean onset of lactogenesis II for women with a BMI ≥ 30 was 85.2 hr postpartum, whereas the cutoff for DLII commonly used is 72 hr. We also found increasing age to be associated with DLII. Women are increasingly delaying childbearing, and the age at which women have their first child has increased over the years (Mathews & Hamilton, 2016). As obesity has become more prevalent, we have an older and heavier pregnancy population. The expectations for the onset of lactogenesis may need to be adjusted so that patients have a realistic expectation as to its occurrence.

A few studies indicate some possible physiologic reasons for breastfeeding difficulties in women with an overweight or obese BMI. Overweight and obese women have been noted to have a decrease in prolactin response to suckling (Babendure et al., 2015; Rasmussen & Kjolhede, 2004). Insulin resistance may delay the time it takes to reach the concentration necessary for the onset of mature milk production. Obesity is also known to increase insulin resistance. Emerging evidence suggests that insulin is necessary for secretory activation and mature milk production (Nommsen-Rivers, 2016). This may therefore also interfere with the pathway to lactogenesis.

These data add support to the already known adverse effects of obesity in pregnant women. Women who start out the pregnancy overweight or obese are at greater risk of excessive weight gain in pregnancy. Clinicians should offer counseling and support to help achieve weight gain in alignment with the Institute of Medicine recommendations. Women with

Table 3. Logistic Regression of Factors Predicting Delayed Lactogenesis II.

Factor	*B*	Wald χ^2	*p*	Adjusted *OR*	95% CI
Age	.11	8.39	.004	1.11	[1.04, 1.20]
Prepregnancy BMI	.06	6.19	.013	1.07	[1.01, 1.12]
Weight gain	.04	8.57	.003	1.04	[1.01, 1.07]
Epidural	.95	4.42	.035	2.60	[1.07, 6.35]

Note. Model $\chi^2 = 26.519$, $p < .001$. *OR* = odds ratio; CI = confidence interval; BMI = body mass index.

a BMI > 30 should gain 11 to 20 lb; with a BMI from 25 to 29.9, 15 to 25 lb; and with a BMI from 18.5 to 24.9, 25 to 35 lb (American College of Obstetricians and Gynecologists, 2013). Breastfeeding counseling should also be individualized to the needs of this population. Anticipatory guidance can help prepare women for the potential challenges.

Given the greater risk of DLII, the newborn may be at risk of excessive weight loss and require supplementation. During the birth hospitalization, in order to protect neonatal gut integrity, donor milk should be considered. These women should have tailored breastfeeding interventions and support. During the birth hospitalization, it may be warranted to have these mothers initiate pumping with a hospital-grade pump in addition to breastfeeding their infant on demand to facilitate the establishment of lactogenesis II and the ability to supplement the infant with expressed milk. Mothers should be taught the critical importance of establishing milk supply and should be encouraged to focus on breastfeeding/pumping and taking care of themselves. Mothers should be taught to monitor their infant's stool and urine output daily. If supplementation is required, mothers should be encouraged to view this as a time limited intervention. Furthermore, appropriate volumes of supplementation should be provided per the Academy of Breastfeeding Medicine clinical protocol.

Postdischarge from the birth hospital, these mothers should receive follow-up lactation support and care within 48 hr. Home visitation or office visits with a health professional trained in lactation are warranted. Mothers may need to continue to pump at home to establish milk supply with the goal of achieving full direct breastfeeding with a normal milk supply.

Strengths and Limitations

The main strength of the study is the prospective design and the close follow-up postpartum until lactogenesis II occurred. This should create an accurate assessment of the onset of lactogenesis II. Another strength is the racially and ethnically diverse sample similar to that of the population of the United States. This improves the generalizability of the findings.

When considering our findings, some limitations should be considered. We used the maternal self-reported onset of lactogenesis II. Although the onset of lactogenesis II by maternal perception is a subjective assessment, it has been validated in previous research and has been associated with excess neonatal weight loss (Brownell et al., 2012; Chapman & Pérez-Escamilla, 2000; Matias et al., 2014). Also, similar to other observational studies, self-selection bias cannot be excluded. In addition, maternal prepregnancy weight in the prenatal record is documented as patient self-report. Evidence suggests that women tend to underestimate their weight. This may have led to an underestimate of prepregnancy BMI (Shin, Chung, Weatherspoon, & Song, 2014).

Conclusion

Although our study found only a small relationship of prepregnancy obesity to DLII, nearly one in four women in the United States begin pregnancy with a BMI ≥ 30; the implications of an association may be of clinical significance (Osterman et al., 2013). As we work to increase breastfeeding rates among these women, the physiologic mechanisms should be considered as potential targets for intervention. Women with these risk factors may benefit from additional education and support both prenatally and postpartum. Future studies are needed to further assess the appropriate amount of gestational weight gain in women to optimize pregnancy and breastfeeding outcomes.

Declaration of Conflicting Interests

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