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Associations between lactation, maternal carbohydrate metabolism, and cardiovascular health

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Introduction

In mammalian reproductive physiology, lactation follows pregnancy. Meeting the nutritional needs of the infant imposes a substantial burden on maternal physiology, requiring about 500 calories per day. Growing evidence suggests that premature weaning disrupts this physiology and impacts long-term maternal health. In the postpartum period, mothers who are not breastfeeding have higher glucose and insulin levels, and lower disposition index, compared with lactating mothers. Long term, never or curtailed breastfeeding is associated with maternal health risks, including higher rates of hypertension, diabetes, and myocardial infarction. These data suggest that maternal lactation may be a modifiable risk factor for long-term maternal health. This manuscript reviews the physiology of lactation, discusses short-term associations between current lactation and cardiometabolic risk markers, and reviews observational data linking lactation with maternal health outcomes. Hypothesized mechanisms are also discussed, including the potential for confounding by maternal health behaviors and preexisting metabolic disease. Finally, evidence-based clinical recommendations are reviewed that optimize a woman's chances of achieving her breastfeeding goals.

Physiology of lactation

Lactation is a two-person organ system, depending on the integrated neurobehavioral dynamics of mother and infant. These dynamics begin during the mother's adolescence, when cyclic stimulation by estrogen and progesterone facilitates development of the breast ducts. During pregnancy, estrogen, progesterone, insulin, cortisol and thyroid hormone all contribute to the elaboration of glandular tissue. By 20 weeks' gestation, the maternal breast is capable of milk synthesis, as indexed by the presence of lactose in maternal urine[1]. After delivery of the placenta, falling progesterone levels are thought to trigger onset of secretory activation, marked clinically by milk "coming in" as gap junctions between lactocytes close, trapping lactose and water in the alveolar lumen. The hormone prolactin stimulates milk synthesis, while oxytocin from the posterior pituitary triggers milk secretion. Oxytocin causes contraction of myoepithelial cells surrounding alveoli in the breast, allowing transfer

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of the milk through the ducts to the areola. At the breast, the infant's oromotor organization determines whether milk is successfully transferred. Latch and milk transfer require mature infant suck-swallow-breath function. In addition, to establish and sustain lactation, mothers must learn to identify and respond to infant feeding cues.

The synthesis of milk depends on availability of substrate and on both endocrine and autocrine regulation. In early lactation, endocrine factors appear to predominate; prolactin levels are highest in the early weeks of breastfeeding. Thyroxin, growth hormone, cortisol and insulin also contribute to normal milk synthesis. Recent evidence from the milk transcriptome suggests that insulin signaling plays a key role in milk synthesis. Among women with insulin resistance (indexed by HOMA) and low milk supply, Lemay et al found increased expression of PTPRF, which interferes with insulin-receptor B signaling and thereby may inhibit milk synthesis[2]. Milk synthesis mobilizes maternal energy stores: lactating women require about 500 kcal per day to produce milk to meet the needs of an exclusively breastfed infant[3]. Evidence suggests that in well-nourished women, the majority of energy from milk production is derived from dietary intake; however, modest calorie restriction does not adversely affect milk supply. In a clinical trial of weight loss during lactation among overweight women, dietary restriction of about 500 kcal a day did not adverse affect infant growth[4].

Lactation and short-term markers of metabolic health

Conventional wisdom holds that breastfeeding helps women to lose weight. Lactation mobilizes about 500 kcal per day, roughly equivalent to 45 minutes of running at a 6 mile-per-hour pace. Evidence from observational studies suggests that longer, more intensive breastfeeding is associated with greater weight loss after pregnancy. Dewey et al prospectively followed 46 breastfeeding women and 39 women who weaned by 3 months; they found that breastfeeding women had 2 kg more weight loss in the first year than the non-breastfeeding women[5]. In the Danish National Birth Cohort, greater breastfeeding duration and intensity were associated with reduced retained gestational weight gain: Women who gained 12 kg during pregnancy and fully breastfed for 6 months were below their pregravid weight by 6 months postpartum, whereas women who breastfed less than 1 week were nearly 2 kg over their pregravid weight[6]. Other studies have not found long-term differences in retained weight: Ohlin and Rossner found that overall weight loss from 2.5 to 12 months was similar, regardless of breastfeeding status. In a subsequent analysis[7], the authors found that women who snacked 3 or more times a day did not lose weight with lactation, suggesting that relatively small differences in dietary habits can counteract effects of lactation on maternal weight.

Only one randomized controlled trial has quantified the effect of lactation intensity on maternal weight. Dewey et al randomized 141 exclusively breastfeeding mother-infant dyads in Honduras to continued exclusive breastfeeding from 4 to 6 months vs. introduction of complementary foods[8]. Women who continued to exclusively breastfeed lost 600 g more during the two-month period than women whose infants received complementary foods. Interestingly, the authors estimated that the exclusively breastfeeding mothers expended an additional 5520 kcal during this two-month period; 600g of fat, at 9 kcal/g, would provide

5400 kcal, meeting almost all of this excess energy need. While these results provide randomized control trial evidence that greater lactation intensity increases weight loss, the generalizability of these data is unclear in high-income countries where nutrient-dense food is abundant.

Several authors have found that lactation is associated with differences in glucose and insulin homeostasis among women with recent gestational diabetes. Kjos et al measured glucose and insulin levels among 809 primarily Latina women with recent gestational diabetes. At 4 to 12 weeks postpartum, non-lactating women had higher fasting and 2-hour post-load glucose values[9]. More recently, Gunderson et al measured glucose tolerance as a function of breastfeeding intensity at 6–9 weeks postpartum and found that exclusively or mostly formula-feeding mothers more likely to have impaired glucose tolerance than exclusively breastfeeding mothers (41.5 vs 24.6%, p=0.02). This association was also present in an analysis restricted to obese women (53.1 vs. 26.2%, p=0.03). Authors reported similar findings in the Atlantic Diabetes in Pregnancy cohort: compared with formula feeding women, women with recent gestational dysglycemia who were currently breastfeeding had an MV-adjusted 0.42 (0.20–0.89) odds of dysglycemia at 12 weeks postpartum[10]. Gunderson further quantified the acute effect of breastfeeding on glucose and insulin metabolism by comparing fasting and 2-hour post-load glucose values among lactating women who did or did not breastfeed during the oral glucose tolerance test. Fasting glucose and insulin values were similar in the two groups, but post-load levels were lower among women who breastfed during the test (MV-adjusted predicted values for glucose 108.8 vs 115.0 mg/dL) suggesting an acute effect of breastfeeding on maternal insulin and glucose homeostasis[11].

Lactation is also associated with differences in lipid homeostasis. Darmady and Postle[12] followed 34 women from prior to conception through pregnancy and the postpartum period and found that triglyceride levels retuned to baseline 13 weeks earlier in lactating than in non-lactating women ($p < 0.001$). Other authors have identified a protective association between lactation and HDL metabolism at six weeks[9,13#565] and 3 months[14] postpartum. In women who continue breastfeeding for 1 year, higher levels of HDL appear to persist until weaning[15]. In the Kaiser SWIFT cohort, greater breastfeeding intensity was associated with higher HDL and lower LDL[16] at 6 to 9 weeks postpartum.

Finally, lactation is associated with lower blood pressure in the postpartum period. The hormone oxytocin plays a central role in milk ejection via its effects on myoepithelial cells, and oxytocin modulates in vascular endothelial function[17]. In animal studies, Petersson et al.[18,19] found that oxytocin administration decreased blood pressure in rats, with effects that persisted for 10 to 21 days after drug administration was discontinued. Observational studies suggest that nursing mothers have diminished autonomic responses to stressors. Mezzacappa et al[20] found evidence that exclusive breastfeeding was associated with lower heart rates, increased vagal tone, and decreased sympathetic response to laboratory stress, compared with values in non-exclusive breast feeders and non-postpartum controls.

Lactation and long-term maternal cardiometabolic health

In observational studies, authors have reported lasting associations between lactation duration and maternal health outcome. Longer lifetime breastfeeding duration is associated with reduced maternal obesity. In a longitudinal cohort in La Crosse, Wisconsin, Rooney and Schauberger[21] found long-term differences in adiposity by breastfeeding history. A decade after the index pregnancy, they reported that women who breastfed for more than 12 weeks weighed 3.7 kg less than women who had never breastfed, adjusting for gestational weight gain, weight loss by 6 months postpartum, and aerobic exercise. In the UK Million Women's Study, Bobrow et al measured associations between breastfeeding duration per child and body mass index at a mean age of 57 years[22]. Each 6-months of breastfeeding was associated with a 0.22 kg/m2 decrease in body mass index.

Longer lifetime lactation is also associated with lower risk of Type 2 Diabetes. In the Nurses' Health Studies, each year of lifetime lactation was associated with a 15% reduction in incident Type 2 Diabetes during the 15 years following a women's last birth[23]. Lifetime lactation was similarly associated with lower prevalent diabetes in the Women's Health Initiative[24]. In a longitudinal cohort study among Kaiser patients, never breastfeeding was associated with a 1.4-fold risk of Type 2 diabetes, compared with breastfeeding for at least 6 months per child[25]. Authors have reported similar associations between lifetime lactation and Type 2 Diabetes in cohorts in China[26], Australia[27], Norway[28], and Europe[29]. A recent meta-analysis found a non-linear association between lifetime lactation duration and incident type 2 diabetes risk[30].

The inverse association between lactation and incident type 2 diabetes has been found in some cohorts of women with Gestational Diabetes. In the Nurses' Health Study II, lactation duration was marginally associated with reduced incident Type 2 Diabetes in unadjusted models (HR per year 0.90, 95% CI 0.81–1.01, p for trend = 0.05), but not in adjusted models[23]. However, in German prospective cohort study of women with GDM who were islet-cell antibody negative[31], more than 3 months of lactation was associated with a marked reduction in progression to type 2 diabetes (MV-adjusted HR 0.54, 95% CI 0.34–0.85) during 19 years of follow-up. In a Canadian study, women with a history of gestational diabetes who had breastfed for >10 months had more favorably glucose homeostasis, indexed by more favorable fasting insulin concentrations and insulin sensitivity indices[32].

Lactation is also associated with differences in hypertension risk. In a Korean cohort study, Lee et al found that never-breastfeeding was associated with an increased risk of premenopausal hypertension[33]. Schwarz similarly found that shorter lifetime lactation was associated with prevalent hypertension in the Women's Health Initiative[24], and Stuebe et al found that women who did not breastfeed had a 1.16-fold risk of incident hypertension, compared with women who breastfed for at least 12 months per child[34]. If this association is causal, the authors estimated that 12% (95% CI 4.9–19) of incident hypertension could be attributed to suboptimal lactation.

Lifetime lactation is also associated with lower risk of metabolic syndrome. Ram et al found that each year of lifetime lactation was associated with a 0.88-fold reduction in risk of

metabolic syndrome (95% CI 0.77–0.99). Gunderson et al quantified associations with metabolic syndrome in the Cardia cohort and found that longer lactation was associated with reduced risk. This difference was more pronounced among women with a history of gestational diabetes. Among women with GDM who breastfed for a lifetime total of > 9 months, the odds ratio for metabolic syndrome was 0.14 (95% CI 0.04–0.55) compared with having breastfed for 0–1 month lifetime. Among women without a history of GDM, this odds ratio was 0.44 (95% CI −.23–0.84). These results suggest that breastfeeding is a powerful modifiable risk factor for incident metabolic disease among women with pregnancies complicated by GDM.

Maternal lactation history is also associated with differences in incident cardiovascular disease. Among parous women in the Nurses' Health Study, lifetime breastfeeding duration of >23 months was associated with a 0.77 (95% CI 0.62–0.94) multivariate-adjusted hazard ratio of incident myocardial infarction, compared with never having breastfed. This association was stronger in the 30 year after a woman's last birth: >23 months of lifetime lactation was associated with a 0.66 (95% CI 0.49–.89) multivariate-adjusted hazard ratio, compared with never having breastfed. Among parous women in the Women's Health Initiative, Schwarz et al found that a lifetime breastfeeding duration of >23 months was associated with a multivariate-adjusted OR of 0.86 (95% CI 0.89–0.98) for prevalent cardiovascular disease, compared with never having breastfed.

A single randomized controlled trial has evaluated the association between an intervention to increase breastfeeding duration and intensity and maternal health[35]. The PROBIT study was a cluster-randomized trial conducted in Repblic of Belarus from 1996 to 1997. The study randomized 34 hospitals to implement maternity practices that increase breastfeeding or to provide usual care. A total of 17,046 mothers-infant dyads, all of whom initiated breastfeeding, were enrolled. The intervention achieved differences in breastfeeding duration: At one year, 19.7% of intervention dyads vs 11.4% of control dyads were still breastfeeding[36]. To determine whether the intervention impacted maternal health, mothers from the PROBIT underwent evaluation of weight, adiposity and blood pressure at 11.5 years postpartum. In an intent-to-treat analysis, allocation to the intervention was associated with a non-significant reduction in obesity (cluster-adjusted OR 0.90, 95% CI 0.71–1.13), body fat ≥40% (cluster-adjusted OR 0.84, 95% CI 0.70,1.01), and hypertension (cluster-adjusted OR 0.85, 95% CI 0.64, 1.12). The study authors concluded that interventions to promote a longer duration of exclusive breastfeeding are unlikely to confer important long-term benefits for maternal adiposity or blood pressure. However, the lack of important differences in the PROBIT sample may reflect the relatively modest impact of the PROBIT intervention on long-term breastfeeding, rather than a lack of effect of breastfeeding on maternal health. PROBIT women in the control group were more likely to discontinue breastfeeding in the first 3 months, compared with women allocated to the intervention (40% vs. 27%). Similar proportions of women stopped breastfeeding between 3 and 6 months (37 vs. 36%), and differences at 6 to 12 months (16 vs. 13%) and 12+ months (20 vs. 11%) were small. Applying the covariate-adjusted hazard ratios for incident hypertension from the Nurses Health Study[34] to the differences breastfeeding rates observed in PROBIT, one would predict an odds ratio for hypertension in the intervention vs. control groups of 0.90, which is similar to the differences found in the analysis. This exercise suggests that

interventions that affect breastfeeding in the first 3 months after birth do not impact long-term maternal health. To determine a causal association between breastfeeding and maternal health, interventions are needed that substantially impact breastfeeding rates from 6 to 12 months postpartum and beyond.

Potential Mechanisms

Several potential mechanisms may mediate the association between lactation and maternal metabolic wellbeing:

1. Lactation may "reset" maternal metabolism after pregnancy
2. Lactation may improve beta cell function
3. Lactation may suppress HPA-axis activity through the action of oxytocin and other lactogenic hormones
4. Lactation may be a marker for other maternal health behaviors
5. Underlying metabolic vulnerabilities may adversely affect breastfeeding

The Reset Hypothesis

Lactation imposes a substantial metabolic burden on mothers, and this mobilization of fat stores may "reset" maternal metabolism after birth[37]. From an evolutionary standpoint, lactation provides a reliable source of calories in the setting of uncertain resources[38]. Mammals accumulate fat stores during pregnancy in anticipation of mobilizing these stores for the infant after birth. If a mother does not lactate, these energy stores are retained, potentially increasing metabolic disease risk. Interestingly, maternal energy balance appears to be closely tied to the hypothalamic-pituitary ovarian axis: in a longitudinal study of Toba women in Argentina, maternal energy balance was correlated with c-peptide levels and resumption of menstrual cycles[39]. Animal studies support a causal association between lactation and subsequent adiposity: dams who are separated from their pups after pregnancy have great visceral adipose tissue at 1 and 2 months postpartum than dams who lactated[40]. In another animal model of three pregnancies followed by lactation or non-lactation[41], non-lactated dams had substantially higher percent body fat than lactated or non-mated control animals (16.7% vs. 10.2% for lactated and 11.0% for controls, $p<0.01$). These findings support the hypothesis that not lactating disrupts normal reproductive physiology, with lasting consequences for maternal health.

Beta cell function and lactation

Lactation may also reduce cardiometabolic disease risk through effects on beta-cell function, as recently reviewed by Much et al[42]. During lactation, glucose is diverted into lactocytes to support milk synthesis, lowering circulating glucose levels and reducing the need for insulin secretion. At the same time, lactating women with recent gestational diabetes had improved pancreatic function, compare with women who were formula feeding. McManus et al[43] assessed postpartum metabolic function in 26 Caucasian women (14 lactating, 12 non-lactating) with gestational diabetes. At three months postpartum, there were no significant differences in insulin sensitivity, glucose effectiveness, or visceral fat or subcutaneous fat;

however, the lactating group did have a higher disposition index, indicating more efficient beta cell function, adjusted for insulin resistance. These differences in insulin secretion may reflect the effects of prolactin on beta-cell proliferation. Prolactin down regulates Menin during pregnancy in a mouse model of gestational diabetes, leading to beta-cell proliferation and improved pancreatic function[44]. Human studies finding differences in fasting insulin and insulin sensitivity after weaning at a function of breastfeeding duration[32] suggest that these effects may persist, conferring lasting protection from type 2 diabetes among women with longer breastfeeding durations.

Stress reactivity

Lactation is associated with differences in maternal stress reactivity. Altemus[45] compared responses to an exercise treadmill test among lactating and non-lactating mothers and found lower ACTH and cortisol levels in the lactating group. Some of this buffering of the stress response appears to be dependent on time since the mother last fed: Heinrichs[46] tested ACTH and cortisol responses to a social stressor among 43 lactating women; half were asked to breastfeed before their appointment, and half breastfed immediately before the stressor. Women who breastfed 30 minutes before the stressor had similar levels of ACTH, but reduced levels of salivary cortisol, compared with women who breastfed 100 minutes before the stressor. The buffering affect may also vary between primiparous and multiparous women: Tu[47] found similar cortisol responses to a stressor among breastfeeding and formula-feeding primiparous women, but among multiparous women, the cortisol response was reduced in breastfeeding compared with formula-feeding women. Other studies have found that women who are lactating display greater aggression when confronted, yet experience less physical stress, evidenced by lower systolic and diastolic blood pressure during a confrontation[48].

The hormone oxytocin is thought to modulate HPA axis activity, and prolonged potentiation of oxytocin pathways in lactation may affect vascular function as well. Among postpartum women, higher oxytocin is associated with decreased sympathetic reactivity to stress[49]. Animal studies have found that oxytocin blunts both cardiovascular and neuroendocrine responses to stress and reduces anxiety-like behavior, whereas oxytocin antagonists increase cardiovascular response to stressors[17]. There is tentative evidence that longer lactation is associated with long-term changes in maternal HPA axis activity: longer lifetime breastfeeding history was associated with higher morning cortisol levels among postmenopausal women[50].

Lactation as a marker for other health behaviors

In the US, mothers who breastfeed differ from mothers who formula feed: they are older, wealthier, better educated, less likely to smoke, and more likely to engage in other beneficial health behaviors[51–53]. To the extent that these health behaviors are associated with long-term maternal cardiometabolic health, these health behaviors, rather than breastfeeding itself, may underlie epidemiologic associations between breastfeeding and long-term maternal health. Importantly, epidemiologic studies have adjusted for multiple health behaviors, including current diet, physical activity, and tobacco use, reducing the influence of these confounders.

Nevertheless, unmeasured or residual confounding may explain some of the association between lactation and maternal health.

Differences in outcomes may also reflect the constraints that can prevent women from initiating and sustaining breastfeeding. Women employed in part-time or hourly wage jobs are less likely to have access to paid maternity leave, and early return to work is associated with shorter breastfeeding duration[54]. Moreover, disparities in health care systems may affect breastfeeding rates. For example, a recent analysis found that neighborhood racial composition was associated with provision of evidence-based maternity practices that support successful breastfeeding. Compared with hospitals in neighborhoods with ≤12.2% black residents, hospitals in neighborhoods with >12.2% black residents were less likely to meet 5 of the 10 indicators for recommended maternity care practices[55]. Successful, long-term breastfeeding may be therefore be a marker for access to high-quality health care, rather than a mechanism to reduce risk. However, if the capacity to breastfeed, rather than breastfeeding itself, improves long-term health outcomes for mothers, we must take steps to ensure that all women have the capacity to achieve their infant feeding goals.

Underlying metabolic health and breastfeeding outcomes

It is also plausible that underlying metabolic vulnerabilities affect a woman's ability to initiate and sustain breastfeeding. In animal studies, excess adiposity is associated with poor milk production and failed lactogenesis[56], and among women, higher maternal body mass index is associated with lower initiation and reduced duration of lactation. Baker et al measured duration of lactation among women in Denmark as a function of pregravid BMI and found lower durations of both exclusive and any breastfeeding among overweight and obese women, compared with women with a normal pregravid BMI[57]. These findings are consistent with other studies of populations in the US[58,59] and Australia[60,61], as well as two recent meta-analyses[62,63]. While psychosocial factors appear to explain the association with *total* breastfeeding duration[64] in a US study, these factors did not explain the association between maternal BMI and *exclusive* breastfeeding, lending support to the hypothesis that greater adiposity impacts a woman's ability to meet 100% of her infant's feeding needs. Authors have similarly found that women with pre-gestational diabetes are less likely to intend to breastfeed than women without diabetes, and women with either gestational or pre-gestational diabetes are less likely to breastfeed in the hospital or to be breastfeeding at hospital discharge[65]. Both GDM and pre-gestational diabetes were also associated with reduced breastfeeding duration in the PRAMS population[66].

Several mechanisms may underlie these associations. In a small study, Rasmussen et al found differences between prolactin levels in normal weight vs. overweight women at baseline and 30 minutes after feeding[67]. Other evidence suggests that both obesity and insulin resistance may play a role in lactogenesis II. Nommsen-Rivers et al found that high BMI was associated with markedly higher rates of delayed onset of lactogenesis[68]: 53.8% of obese women experienced onset of lactogenesis more than 72 hours postpartum, compared with 44.8% of overweight women and 31.4% of normal weight women. In a subsequent study, Nommsen-Rivers further found that maternal metabolic health during pregnancy, indexed by insulin-to-glucose ratio after a 50g glucose load, was correlated with earlier

onset of lactogenesis[69]. Matias et al similarly found a relationship between metabolic health during pregnancy and early breastfeeding outcomes. In the SWIFT cohort of women with gestational diabetes, both prepregnancy obesity and insulin treatment during pregnancy were associated with delayed onset of lactogenesis[70]. These observational findings correlated with recent translational work linking early milk supply problems and insulin resistance: Lemay et al found that supply issues were associated with increased expression of protein tyrosine phosphatase, receptor type, F (PTPRF), a protein that interferes with insulin signaling[2]. These data suggest that underlying insulin resistance may interfere with lactation physiology, supporting the hypothesis that successful breastfeeding may be a marker for metabolic well-being.

Clinical implications

In population studies, never or curtailed breastfeeding is associated with adverse cardiometabolic outcomes. While some of these associations may be explained by confounding behaviors and underlying maternal metabolic disease risk, the existing evidence suggests that enabling women to initiate and sustain breastfeeding can improve long-term health maternal health. Therefore, women's health providers should optimize each a woman's ability to breastfeed her infant[71,72]. Providers should ensure that the outpatient office is conducive to breastfeeding, beginning with provisions for lactating office employees to express milk during the workday. Formula marketing materials should not be present in the health care settings[73]. Breastfeeding should be welcome in waiting areas, and ideally, nursing mothers should be offered the option of a private area to feed if they would feel more comfortable there. Providers and staff should understand the nuances of medication safety in lactation and be a resource for evidence-based advice. Breastfeeding should be discussed in prenatal care and anticipatory guidance provided. Maternity providers should work with hospitals to implement the Ten Steps to Baby Friendly Maternity Care in order to increase each woman's likelihood of achieving her own breastfeeding goals[74].

In the early postpartum period, maternal obesity and insulin resistance impact the management of the breastfed neonate. Infants that are large for gestational age or whose mothers have diabetes are at risk of complications such as hypoglycemia and neonatal jaundice, and monitoring and management protocols should be designed to optimize establishment of breastfeeding. For example, early, prolonged skin-to-skin care after birth increases infant glucose levels: in a Cochrane meta-analysis, continuous skin-to-skin care increased neonatal glucose levels at 60–75 minutes after birth by 10.56 mg/dL[75]. Such skin-to-skin care can be provided during cesarean births, and is associated with reduced formula supplementation during the maternity stay[76]. For infants requiring monitoring for hypoglycemia, ongoing skin-to-skin care and feeding of hand-expressed colostrum or donor human milk can obviate the need to supplement with formula. Similarly, for infants with jaundice, family-centered care plans such as bilirubin blankets can facilitate breastfeeding during treatment[77]. Such provisions are outlined in the Academy of Breastfeeding Medicine protocols for hypoglycemia[78] and jaundice[79]. Following hospital discharge, the maternity provider should be a resource for breastfeeding families, working closely with the infant's provider and with lactation consultants to address any breastfeeding challenges.

Several studies have tested interventions to improve breastfeeding outcomes among overweight or obese women, with mixed results. Rasmussen et al tested phone support and early initiation of pumping among 74 women in New York State and found that neither intervention improved breastfeeding outcomes[80]. Chapman et al tested a peer counselor intervention tailored to the perceived needs of overweight and obese women among 206 women in Connecticut and found higher rates of any and >50% breastfeeding at 2 weeks postpartum, but no other differences in breastfeeding rates[81]. Carlsen et al tested an IBCLC phone support intervention among 226 obese women in Denmark and increased both any and exclusive breastfeeding duration[82]. Carlsen's findings suggest that targeted support may improve outcomes among women at metabolic risk, but it remains to be determined what strategy may be most effective within the United States.

Conclusion

Among parous women, longer lactation is associated with lower risks of diabetes, hypertension, metabolic syndrome, and myocardial infarction. Several mechanisms may mediate these associations; underlying differences in health behavior and health status between women who decide to breastfed and women who decide not to breastfeed likely play a role. However, evidence suggests that enabling a woman to initiate and sustain breastfeeding has the potential to reduce her lifetime risk of cardiometabolic disease. Women's health providers should therefore make every effort to share this evidence with women to inform their feeding decision, and to provide evidence-based care that optimizes each woman's likelihood of achieving her breastfeeding goals.

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